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THE CALORIC REQUIREMENTS OF NORMAL INFANTS AND CHILDREN FROM BIRTH TO PUBERTY

FRITZ B. TALBOT, M.D.
BOSTON

During the past eight years Dr. Francis G. Benedict and I have been conducting a series of studies at the Nutrition Laboratory of the Carnegie Institution of Washington on the energy metabolism of normal infants and children from birth to puberty. A preliminary report of these investigations was recently given by Dr. Benedict in the Shattuck Lecture delivered before the Massachusetts Medical Society at Boston, June 3, 1919.[1] The detailed report of these observations will be published later in a publication of the Carnegie Institution of Washington and will contain a review of the literature of the metabolism of normal infants and children, and a complete discussion of the normality of the subjects studied. In this series of investigations we included 108 boys and 70 girls. The majority of the younger subjects were normal breast-fed infants. These were studied at the Boston Directory for Wet-Nurses. The older subjects were selected from the New England Home for Little Wanderers, all possible efforts being directed towards individuals who represented normality.

Photographs are shown and a description of the apparatus and technic used in these investigations is given in detail in the article by Benedict.[1]

Chart 1 gives the total basal metabolism for twenty-four hours of normal children from birth to puberty. In this instance the calories are plotted against age, showing that the total calories become larger as the age increases. By "basal metabolism" is meant the minimum metabolism necessary to maintain life with the patient at complete muscular repose, and, if possible, without any food in the stomach. It was impossible, however, to study the younger subjects without food. The older subjects were studied in nearly all instances without food.

Chart 1 shows that there is no difference between the basal metabolism of boys and girls up to about one year of age, or about 10 kg. of weight, after which there is a sex differentiation, boys of the same weight having a somewhat higher metabolism than girls of the same weight until they reach 34 kg. of body weight, when the metabolism

1. Benedict, F. G.: Boston M. & S. J. **181**:107 (July 31) 1919.

of both sexes is the same, after which the basal metabolism of girls is somewhat higher than that of boys.

If the basal metabolism shown in Chart 1 were doubled it would approximate the total calories that a child of a given weight would require. This, however, is not quite accurate because the growth factor is greater during the first year of life than it is during later childhood. It is necessary, therefore, to a little more than double the basal metabolism before the age of six months in order to obtain the total metabolism, whereas, after six months of age, doubling the basal metabolism more than gives the total number of calories that are necessary for a child to grow. Whether these figures will be sufficient

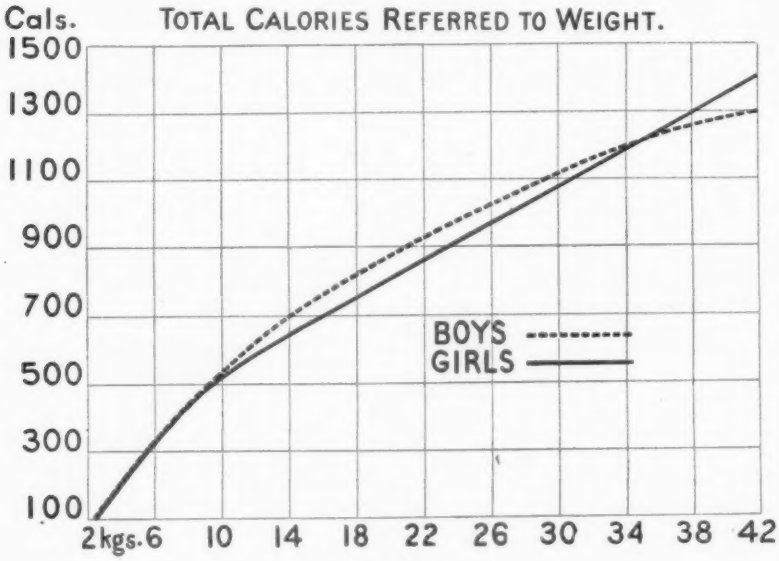

Chart 1

will depend upon the habits of the child, as shown by Captain Gephart at St. Paul's School in Concord, N. H., in which he found that the average intake in food in his subjects was 5,000 calories per day.[2]

Chart 2 gives the measured total basal metabolism of infants from birth to one year, and at the same time estimated curves showing the weight, food intake and available food.

The lower line on Chart 2 represents the total basal metabolism, which, at six months, is 400 calories. This does not mean, however, that all normal children have a basal metabolism of 400 calories at six months of age, since some infants grow much more rapidly and attain a greater length and weight than other infants of the same age.

2. Gephart, F. C.: Boston M. & S. J. **176**:17 (Jan. 4) 1917.

Infants with a greater weight than the average tend to have a higher basal metabolism than the curve given here, while normal infants with less weight than the average usually have a slightly lower basal metabolism.

The upper curve represents the normal increase in body weight of an average infant from birth to one year, and shows the initial loss of

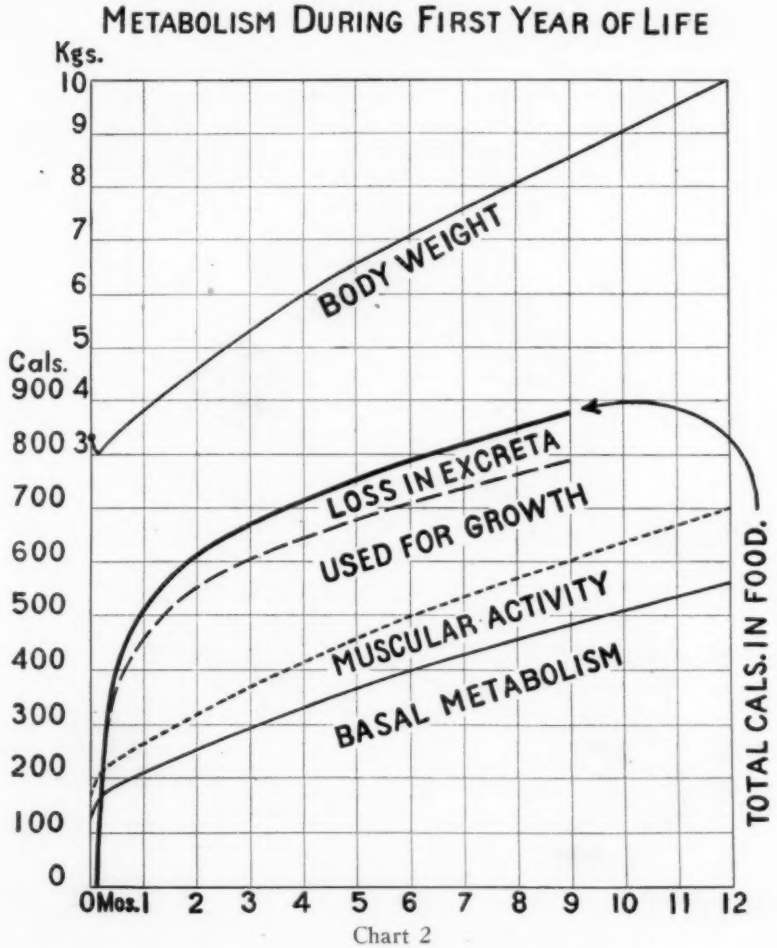

Chart 2

weight which occurs during the first three days of life, before the breast milk "comes in."

The heavy line to which the arrow points indicates the intake of breast milk, which commences about the third day and represents on an average the total calories normally received about this time. This rises rapidly after a sufficient amount of breast milk calories are sup-

plied for the infant to gain in weight. This curve, representing the number of calories in the breast milk, is estimated from figures found in literature. The total number of calories supplied in the food in this manner naturally vary with the different nursing mothers, and are influenced both by the quality and quantity of the milk. It may be

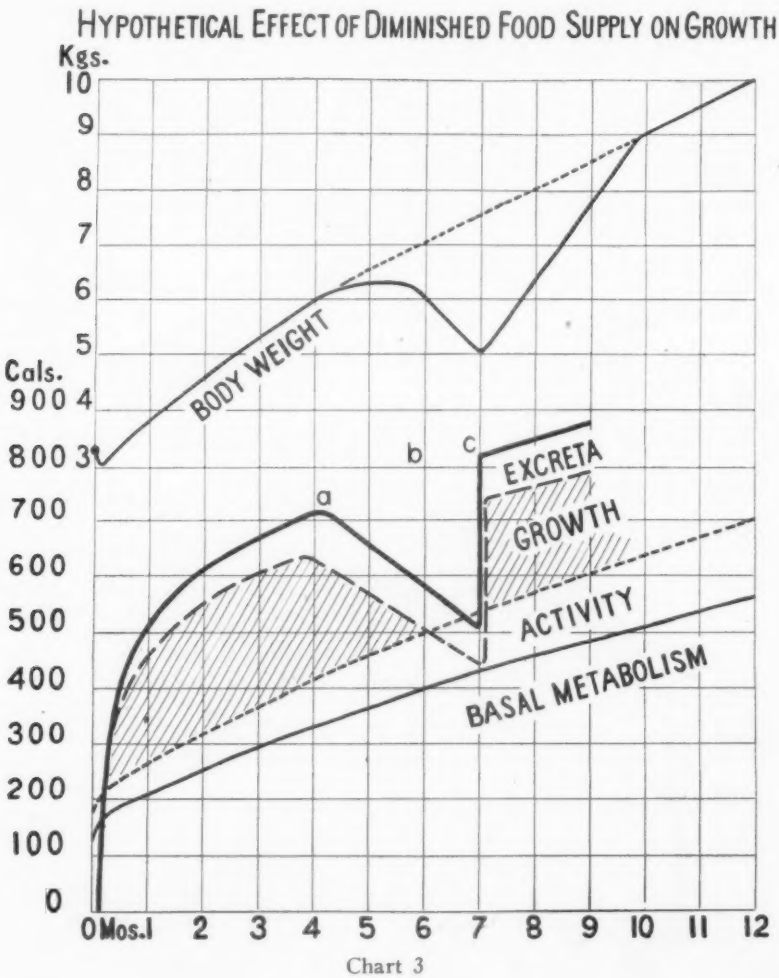

Chart 3

assumed generally that at the end of the first month a baby will be receiving, on an average, approximately 500 calories a day, and at the end of the ninth month about 880 calories a day.

It has been estimated that about 90 per cent. of the food taken by an infant is available for use in the body, the balance being lost in the excreta. Taking this as a standard, the curve in Chart 2 represented

by long dashes gives the available energy which an infant may use at a given age. For example, at six months about 785 calories are ingested, about 705 of these calories being available for use in the body, the remaining eighty calories having been lost in the excreta.

It has been shown by other investigators as well as ourselves, that muscular activity increases the metabolism. We have shown that in

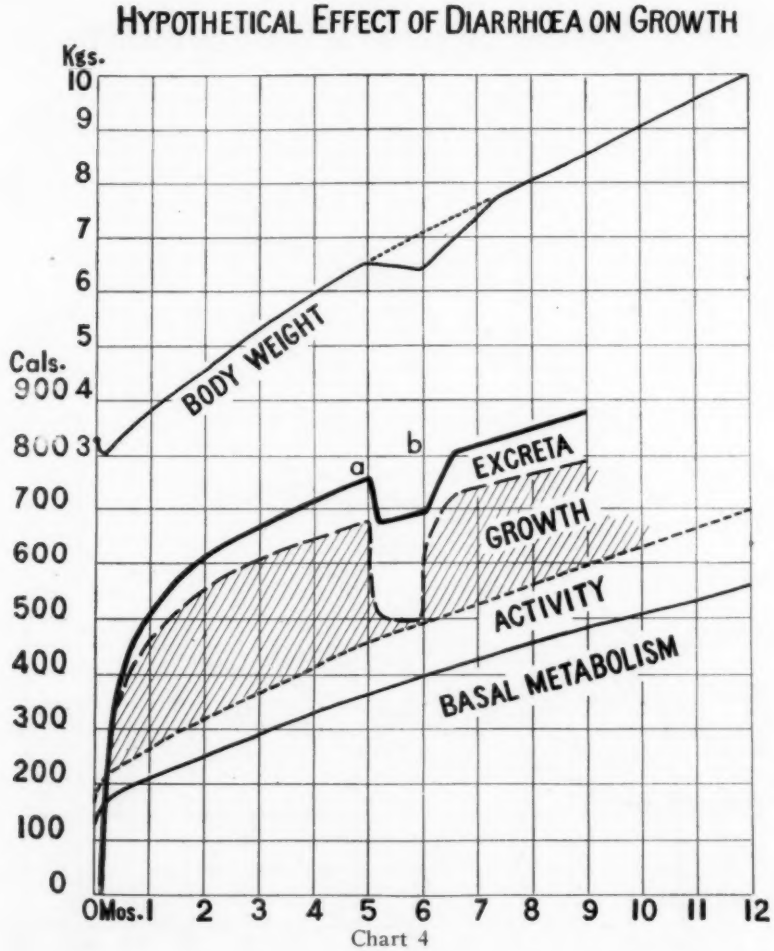

Chart 4

new-born infants crying and kicking may increase the metabolism as much as 200 per cent. for a short time. To estimate the energy used up by muscular activity throughout an entire day, continuous direct measurements are necessary. This was done in two normal infants previously reported by Talbot.[3] In one of these infants the increase

3. Talbot, F. B.: Am. J. Dis. Child. **14**:25 (July) 1917.

in metabolism throughout the twenty-four hours was 30 per cent., and in the other 20 per cent., over the basal metabolism. It may be assumed, therefore, until further figures are obtained, that the average increase of metabolism, due to muscular exercise, over the basal metabolism in a normal infant is 25 per cent. This percentage was, therefore, added to the basal metabolism and is represented in Chart 2 by the

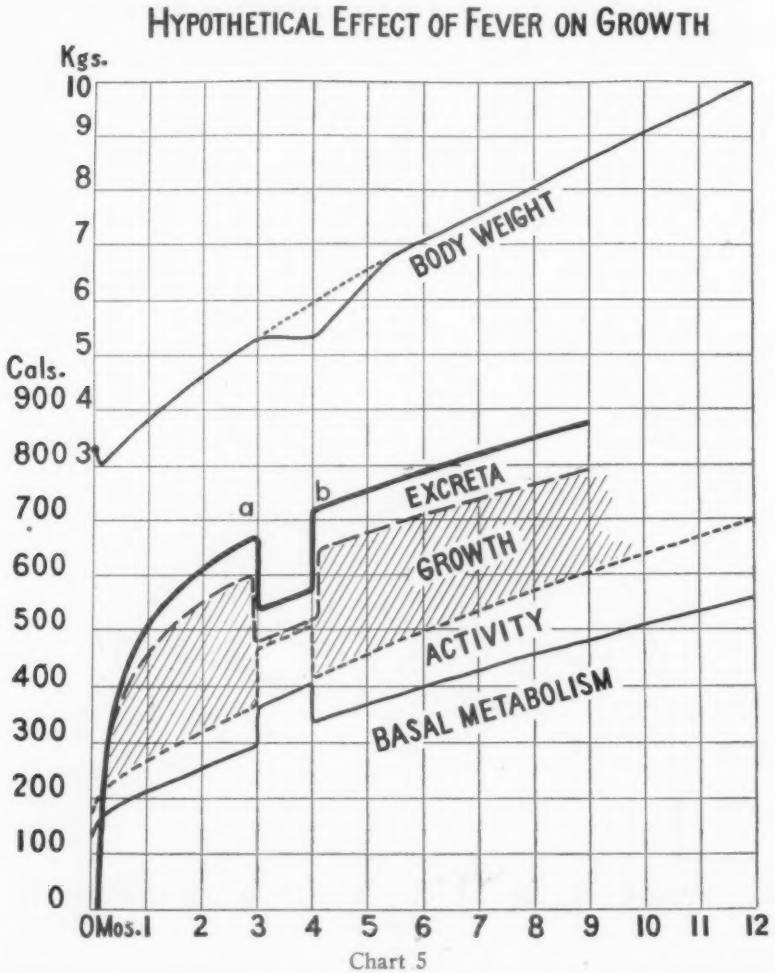

Chart 5

short dashes, and shows that at six months the basal metabolism of an infant is 400 calories with 100 more calories used up during the day in muscular activity. This figure, however, can only be taken as the average amount, since it cannot be assumed that all infants have the same amount of muscular activity.

The greatest rate of growth is during the first five to six months of life when an infant doubles its birth weight. During the second six months of life, the gain in weight is approximately the same as during the first five or six months, but, whereas, the increase in weight was 100 per cent. in the first six months of life, it is in the neighborhood of 50 per cent. in the second six months. Since the period of

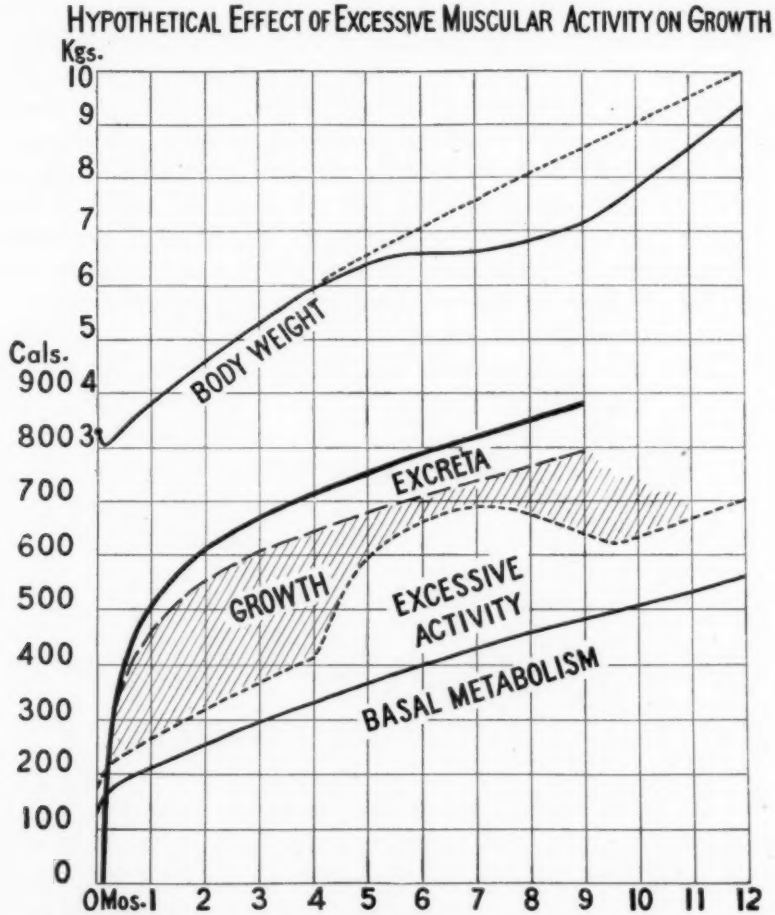

Chart 6

greatest growth is during the first year of life, relatively more food must be available for growth during this period than at any other time. It is obvious that the rate of growth is affected by the number of available calories. In Chart 2 the space between the lines of short and long dashes represents approximately the amount of food which goes for growth. According to the figures on this chart, which, it

must be remembered, are only estimations, 36 per cent. of the food intake went for growth at three months; 26 per cent. at six months, and 21 per cent. at nine months. According to Cammerer,[4] only 15 per cent. of the food energy was retained for growth, while Rubner and Heubner[5] found it to be 12.2 per cent.

In Chart 3 is shown the hypothetical effect of a diminished food supply on growth.

According to Chart 3, an infant received sufficient breast milk for growth up to four months of age, to the point marked *a*, at which time the breast milk began to give out. During the course of the next three months the milk continued to diminish very rapidly, but the mother was unwilling to give a supplementary bottle feeding. At the point marked *b* it is seen that the available energy just equals the calories extended by the basal metabolism and the muscular activity of the infant, and that nothing is left for growth. At this point the infant's weight is stationary. Between the points marked *b* and *c* the available energy is less than the amount used up by the infant in its basal metabolism and muscular activity. At this period the infant must obtain its food from some other source, and does so by using up its body tissue, thus losing in weight from 6 to 5 kg. Presumably, this is done at the expense of its own body fat.[6] At the point marked *c* the condition of the infant becomes so serious that the parents are willing to consult a physician and have a supplementary bottle feeding given. Enough calories are then added to bring the total food up to the normal requirements for growth, the infant gains rapidly, and after enough food is given and properly digested, it reaches the average weight for its age in a short time. Osborne and Mendel[7] have shown by their observations on white rats that it is possible to resume growth after long continued failure to grow.

Chart 4 shows the hypothetical effect of diarrhea on growth.

The recent work of Holt and his co-workers[8] shows that a great deal of fat is lost in the stools during diarrhea. Normally, 91.3 per cent. of the fat in the food is retained in the body, while in diarrhea only 79.3 per cent., and in severe diarrheas only 58.5 per cent. is retained. There is a corresponding loss of protein when casein curds

4. Cammerer: See Lusk, The Science of Nutrition, Ed. 3, Philadelphia and London, W. B. Saunders & Co., 1917, p. 412.

5. Rubner and Heubner: See Lusk, The Science of Nutrition, Ed. 3, Philadelphia and London, W. B. Saunders & Co., 1917, p. 412.

6. Benedict, F. G., and Talbot, F. B.: The Physiology of the New-Born Infant, Carnegie Institution of Washington, Publicatoin No. 233.

7. Osborne, O. T., and Mendel, L. B.: J. Biol. Chem. **22**:439 (Sept.) 1915.

8. Holt, L. E., Courtney, A. M., and Fales, H. M.: Am. J. Dis. Child. **17**: 423 (May) 1919.

are found in the stools. In one instance noted by Talbot and Gamble,[9] 24 per cent. of the ingested nitrogen was lost in the stools.

In Chart 4 it may be assumed that at the point marked *a* an infant commences to have diarrhea, and, as a consequence, there is a great increase in the number of calories lost in the stools, as shown by the chart, with the usual loss in appetite. The available energy is then reduced to such an extent that the infant has nothing left for growth and stops gaining. If the infant becomes restless, and the muscular activity increases, more than 25 per cent. more calories will be burnt up than are available for use, and the infant will lose weight. At the point marked *b* the diarrhea stops, there is no longer excessive loss of energy in the stools, and growth is resumed.

Chart 5 shows the hypothetical effect of fever on growth.

It has been shown that a rise in body temperature increases the metabolism.[10] In Chart 5 it may be assumed that an infant becomes feverish at the point marked *a*, and there is an increase in the basal metabolism. Associated with fever is nearly always an increase in muscular activity and restlessness due to discomfort, with a loss of appetite and consequent drop in the caloric intake. This is shown on the graphic chart and the result is that during fever there is no food left for growth. If the available energy becomes less than the energy used up by the basal metabolism plus the muscular activity the child loses weight.

Chart 6 shows the hypothetical effect of muscular activity on growth.

Northrup[11] reported several cases of infants with nervous prostration in which there was excessive muscular activity due to prolonged mental stimulation. Such infants are continuously on the *"qui vive,"* are very active with the slightest provocation, sleep less than the normal, and practically always lose their appetite. The effect of excessive muscular activity on growth in such cases is illustrated in Chart 6. It also pictures what happens to the older child who overexercises and runs himself "as thin as a rail."

Chart 1 and the lower line on Chart 2, representing the basal metabolism, are the results of actual measurements of the metabolism of normal infants and children. The remaining curves on Chart 2 and Charts 3, 4, 5 and 6 were plotted merely to give a graphic picture of characteristic cases. They do not represent cases actually measured in real life.

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9. Talbot, F. B., and Gamble, J. L.: Am. J. Dis. Child **12**:333 (Oct.) 1916.
10. Lusk: Loc. cit., p. 499.
11. Northrup: Arch. Pediat. **22**:14, 1905.

INFLUENZAL CROUP*

HENRY LOWNDES LYNAH, M.D.
NEW YORK

During the recent pandemic of influenza I had an opportunity to study the laryngeal and tracheobronchial pictures in influenza patients in hospital and private practice. Some of them were supposed to be suffering from diphtheritic laryngeal stenosis, until direct laryngoscopy and bronchoscopy revealed the true nature of their respiratory difficulty.

For some time it has been my routine to make direct laryngeal and bronchoscopic examinations on patients suffering from croup admitted to the Willard Parker Hospital to ascertain the cause of the stenosis. In a number of cases examined during the past year, we were surprised to see that there was no diphtheritic membrane present. At times the larynx was fiery red, and the ventricular bands and vocal cords were edematous and stippled with whitish points on a deep red background. In these cases the edema was supraglottic and interfered with inspiration. In other cases the voice was not lost as in the supraglottic type and the larynx was apparently normal though the obstruction to respiration was extreme. These cases showed the subglottic and tracheal regions to be the chief site of the lesion. In some instances the lesion was confined to the lower trachea and bronchi, and accompanied by symptoms of asthma. In other cases the entire airway was involved.

Apparently, there are four distinct types of respiratory involvement according to the location of the lesion: (a) Laryngeal; (b) tracheobronchial; (c) bronchopulmonary or asthmatic, and (d) pneumonic.

A. The Laryngeal Type.—This type of influenzal croup closely resembles diphtheritic croup, and is indistinguishable clinically unless a direct laryngeal examination is made. Sometimes a feature of influenzal croup is the absence of gradual progressive rhythmic inspiratory and expiratory dyspnea which is frequently present in diphtheritic croup. At times in influenzal croup there is a persistent recurrent glottic spasm which is very distressing to the patient. This may produce a well marked inspiratory crowing sound, and if subglottic stenosis be present there is an expiratory croupy cough. The cough may be the combination of a whoop and a croup. In influenza the

* Read before the Section on Laryngology, Otology and Rhinology at the Seventieth Annual Meeting of the American Medical Association, Atlantic City, N. J., June, 1919.

larynx and trachea are extremely irritable. The recurrent whoop and paroxysmal cough may even suggest pertussis. The whoop is caused by glottic spasm together with tumefaction of the ventricular bands which tend to close the glottic aperture. These children may become very blue and require intubation. Croup is purely an expiratory phenomenon, and is due to edema and inflammation of the subglottic and tracheal regions. When the stenosis is marked physical examination of the chest may show that no air enters the lungs. Diminished respiratory murmur or a unilateral bronchial obstruction cannot be elicited until after the airway has been made free by the introduction of an intubation tube or bronchoscope.

B. The Tracheobronchial Type.—In these cases there may be a marked tracheobronchitis for several days before definite signs of respiratory difficulty or a croupy cough develops. In these cases the voice may be normal and the child may cry aloud though dangerous obstructive symptoms are present. Intubation may be necessary to save life, because of extensive subglottic and tracheal edema. The larynx usually appears normal, but when a tracheoscope or bronchoscope is introduced into the trachea, the latter is seen to be fiery red and edematous. When the edematous flaccid posterior tracheal wall comes in contact with the anterior edematous mucosa the tracheal lumen and expulsive cough may be shut off almost completely. The trachea and bronchi may present a stippled appearance with scattered adherent plaques of tenacious mucus which may be blood streaked. Collections of gluelike mucus may completely obstruct a bronchus, and in the moderately severe types aspiration of the tenacious secretion may induce recovery if there be no lung involvement.

C. The Bronchopulmonary or Asthmatic Type.—In these cases the onset may be extremely violent or the condition may be ushered in with all the premonitory symptoms of laryngotracheobronchitis. The child may be stricken with a fulminating influenzal asthma even while asleep. This may be mistaken for some form of food anaphylaxis, and the history may bear this out if we are told that the child went to bed in good health aside from a slight cold in the head. The lungs are enormously engorged with blood, and there is extreme difficulty with respiration. Violent glottic spasm may be present and add to the gravity of the picture. The cough may be lost completely. These patients rasp and wheeze and are profoundly prostrated. There is a short stridulous inspiration and a prolonged wheezing expiration audible at some distance. On introducing the bronchoscope we may be surprised to find very congested bronchi comparatively free from secretion. A few streaks of tenacious bloody mucus may be present. As the secretion is poured out the pulmonary congestion decreases.

Many of these children succumb within a few hours after the onset of the attack. When secretion is profuse it may be aspirated bronchoscopically and afford relief. Dr. Jesse G. M. Bullowa agrees with me in the belief that marked spasm of the bronchi is not the cause of the asthmatic attack. Spasm is not seen bronchoscopically. That the cause is probably due to the enormous engorgement of the lung producing inelasticity. The markedly engorged inelastic lung is not expanded on inspiration so that the functionating elastic vesicles are overdistended, producing acute pulmonary emphysema. If the condition becomes extreme, interstitial or tissue emphysema may result. In this type of case the absence of cough may be noteworthy, even on the introduction of the bronchoscope. These children may be able to cry aloud at times. Their difficulty is expiratory. Epinephrin hypodermically may afford relief. Finally, (d) there may be the lobar or bronchopneumonic types of dyspnea, with characteristic air hunger.

The following cases are some of those studied during the epidemic.

REPORT OF CASES

CASE 1.—A girl, 4 years of age, had a persistent croupy cough which came on four days after an ordinary catarrhal cold and slight bronchitis. The croupy cough continued to grow worse the following day and a dose of antitoxin was administered. As the croup did not improve within the next twenty-four hours, and as a marked glottic spasm was an added complication, the late Dr. Ralph Whitcher asked me to determine the cause of the laryngeal obstruction by direct observation. The larynx was slightly reddened. The aryepiglottic folds and ventricular bands were stippled. The cords were red. There was a moderate degree of subglottic edema (Plate I, Fig. 3). On the introduction of the direct speculum the glottic spasm was greatly increased. A 5 mm. bronchoscope was promptly introduced which opened the glottic aperture and gave immediate relief to inspiration. The tube, however, met with slight resistance in the subglottic region which was edematous. The tracheobronchial lesion was studied for three minutes. The trachea was red and stippled like the larynx, there was little bronchial secretion (Plate 2, Fig. 3). There was no evidence of diphtheritic membrane. Suction cultures aspirated into a sterile bottle were reported by Miss Valentine as showing pneumococci and a few short rods resembling influenza bacilli. No diphtheria bacilli were present.

After the removal of the bronchoscope the child breathed easily, and the slight amount of dilatation of the subglottic region greatly improved the croupy cough. The following day the child was greatly improved, and aside from an occasional mild whoop she seemed on the road to recovery. The expiratory croupy cough had practically disappeared. The case was one of laryngotracheobronchitis without pulmonary involvement.

CASE 2.—Male infant, 11 months old, was apparently ill with an ordinary cold on the chest. There were a few scattered râles over both lungs. His four years old sister had suffered the week before with croup and had received diphtheria antitoxin though the cultures from the nose and throat were negative. Immunizing doses of antitoxin were given at that time to the other children in the family. A few days later the baby became very dyspneic and was given at this time a curative dose of antitoxin. Three days later the croupy symptoms became extreme, and it was at this time that I was called by Dr. Whitcher to make a direct laryngeal examination, for he suspected influenzal

laryngeal edema as being the probable cause of the difficulty on account of the absence of response to antitoxin.

The physical signs in the chest at this time revealed a diffuse bronchitis though very little air was entering the lungs. The temperature was 102.4 F., the pulse was uncountable, respiration was labored. A rapid direct examination showed only a slight reddened larynx. The vocal cords were apparently normal (Plate 1, Fig. 4). The dyspnea was urgent and the cyanosis marked. A 4 mm. bronchoscope was introduced immediately between the apparently normal cords. The subglottic and tracheal regions were very edematous and the tube was introduced through a ring of annular edema with considerable difficulty. There was no evidence of diphtheritic membrane. The trachea and both main bronchi were very red and there were many scattered patches of mucopus on the walls, which were aspirated into a sterile bottle for examination. Miss Valentine reported streptococci in abundance, pneumococci and influenza bacilli.

The infant was able to breathe comfortably through the bronchoscope. Five minutes after its removal intubation became necessary. A small flat headed bulbous noncough-up tube was introduced. Following the examination the temperature rose to 103 F., but no definite signs of pneumonia could be elicited.

There was a marked tracheobronchitis and many râles produced by the rattle of the mucus in the intubation tube. On the fourth day after intubation the temperature fell to 100 F. and the tracheobronchitis was greatly improved. The intubation tube was removed on this day but within an hour, because of subglottic edema, it was replaced. Because of the reaction, cough and increased tracheobronchitis no further attempt was made to remove the tube for six days. On the second trial the persistent subglottic edematous stenosis necessitated the reintroduction of the tube within thirty minutes. Glottic spasm also added to the discomfort during this short period of detubation. The general condition of the child gradually returned to normal three days after the second detubatory trial. We then decided to leave the tube in for one week before the next trial removal. This time on removing the tube the 4 mm. bronchoscope was immediately introduced with ease. It was left in for a few minutes and then gradually withdrawn. The infant was able to breathe fairly well after the removal of the bronchoscope, and made a complete recovery.

CASE 3.—A boy, 10 years old, had been in perfect health until he became overheated by skating. That night he was seized with violent dyspnea and asthmatic breathing. Dr. Isadore Friesner recognized the existence of an obstruction in the larynx or trachea below the cords, because the boy was able to speak with a normal voice when the respiratory difficulty temporarily improved. Dr. Friesner desired a direct laryngeal and bronchoscopic examination to determine the cause of the obstruction.

By physical examination of the chest it was found that much less air entered the right lung than the left. These findings were confirmed by Dr. Koplik. There was slight cyanosis.

On direct examination the larynx was slightly cyanotic but otherwise normal in appearance. The vocal cords and ventricular bands were not swollen. There was marked subglottic edema which looked dark and grayish (Plate 1, Fig. 1). A mass of tenacious mucus clung to the interarytenoid space. The 5 mm. bronchoscope was introduced with moderate difficulty on account of the firm subglottic edema. The urgent dyspnea immediately disappeared, but a slight asthmatic wheeze persisted though the bronchoscope had reached the carina of the trachea. There was no evidence of diphtheritic membrane. The trachea had a purplish red color, and there was a thick glue-like exudate covering the posterior wall. The carina was somewhat dusky with a thick tenacious blood stained plaque of mucus on the posterior wall (Plate 2, Fig. 1). This was aspirated into a sterile bottle and the right bronchus was explored. The right bronchus was almost completely filled with thick tenacious bloody

mucopus, which constantly poured from the superior, middle and inferior branches (Plate 2, Fig. 5). The left main bronchus and its branches were comparatively free from secretion, and there was little sign of bronchial inflammation. All of the secretion was removed by suction and the boy's condition greatly improved.

The examination without anesthesia lasted for five minutes. The cough reflex was not lost. There was a gradual return of the dyspnea, and four hours later a second bronchoscopy was performed and secretion removed with immediate relief. An intubation tube was then introduced to prevent recurrence of subglottic stenosis and in a short time the boy fell asleep.

The following day the general condition became worse and we again bronchoscoped him. There was little or no improvement though the secretion was aspirated. The right main bronchus was beefy red and edematous. The temperature was 104.3 F., respirations 52 and pulse 100. The marked cyanosis was only slightly relieved by bronchoscopy and intubation. The cough reflex was lost, and the boy was unable to expel the rapidly forming secretion. He sank rapidly on the third day of his illness. He was delirious, cyanosed and unable to cough. The respirations were labored and the lungs were filled with bubbling râles. The urine was bloody and scant. Twenty-four hours later he succumbed to massive pulmonary involvement. The suction cultures taken from the tracheobronchial tree showed *Streptococcus hemolyticus* in abundance.

Case 4.—A boy, 3 years of age, ill three days, was admitted to the Willard Parker Hospital suffering from laryngeal diphtheria. The child was very croupy and a direct laryngeal examination disclosed a membrane on the ventricular bands and vocal cords. Dr. Thompson intubated the patient shortly afterward. A dose of 10,000 units of diphtheria antitoxin was given intravenously. Direct cultures, taken just before intubation, showed diphtheria bacilli. The intubation tube was worn for four days and then removed. The boy remained without the tube, and after having had a series of negative cultures from the nose and throat was ordered for discharge.

On the day set, the temperature suddenly rose to 104.8 F., and there was a rapid return of the croup, for which condition he had been admitted. It was first thought that the recurrent croup was due to subglottic stenosis following diphtheria. However, the fluctuating temperature and diffuse bronchitis suggested influenza, especially in the presence of the epidemic and the numerous cases in the hospital.

A direct examination revealed a beefy red larynx with marked tumefaction of the ventricular bands (Plate 1, Fig. 6). The subglottic region was red and edematous though the 5 mm. bronchoscope was introduced without much difficulty. The trachea and bronchi were reddened and there was a moderate amount of bronchial secretion coming out of the left bronchus (Plate 2, Fig. 4). Suction and swab cultures taken from the larynx, trachea and bronchi showed no diphtheria bacilli, but pneumococci and influenza.

The bronchitis and fluctuating temperature lasted until the end of the fifth week. Reintubation was not necessary after the removal of the bronchoscope, for it had dilated the subglottic area sufficiently to afford relief. This case illustrates the sequence of two different infections each causing acute laryngeal stenosis. The child recovered.

Case 5.—A girl, 6 years of age, was admitted to the Willard Parker Hospital after intubation in her home by the ambulance surgeon. On admission the child was in poor condition. The temperature was 105.2 F., pulse uncountable and respirations 58. She had received 10,000 units of diphtheria antitoxin at the outset of the croup two days previously. Little air entered the lungs in spite of the tube, but because of the desperate condition of restlessness and delirium, it was deemed wise not to disturb her. She seemed moribund. The lungs were filled with moist râles, she was deeply cyanosed like all dying influenza patients.

We were surprised the following day to find the patient still alive and apparently improved. Epinephrin had been administered, but the dyspnea and cyanosis were not completely relieved. By direct laryngoscopy the tube was removed. The pharynx was red with granular white spots on the postpharyngeal wall, and also on the buccal mucosa. There was no diphtheritic membrane present. The larynx was beefy red and stippled with small white points. The trachea was swollen, but there were no apparent hemorrhages. The distended vessels were plainly visible. Thick ropy blood streaked secretion partially obstructed the lower trachea and bronchi. There was no cough on the introduction of the bronchoscope even when the bifurcation of the trachea was reached. There was slight asthma present with the bronchoscope in place. The child was delirious during the manipulation. After aspiration of secretion the extreme cyanosis was slightly relieved. The intubation tube was reintroduced. Cultures taken by suction showed no diphtheria bacilli, pneumococci and a few influenza bacilli.

On the third day after admission the temperature rapidly fell to 100 F., pulse and respiration were unchanged. Five days after admission, the child was still wearing the tube and still delirious. Areas of bronchopneumonia were found in the chest on physical examination. There was slight cyanosis. On the seventh day the tube was removed but replaced in five minutes because of subglottic edema. On the eleventh day after admission the temperature was normal and the child's general condition was good. Dr. Thompson removed the tube and the child remained without it for six days, when she again became dyspneic. The area of pneumonia seemed more extensive on physical examination and the temperature rose to 103 F. Two days later the child was again intubated to relieve a distressing spasm of the glottis and the retention of secretion in the lung. The following day the child coughed up the tube and remained without it thereafter. After a convalescence lasting five weeks she was discharged cured.

CASE 6.—An infant, 11 months of age, was seen in consultation with Dr. Sheitlis, and intubated shortly afterward. The baby had had a slight cold in her head and after a few days in bed was pronounced well. A few days later the coryza recurred, the temperature rose to 103 F., and there was a slight cough. After several days the bronchitis subsided. Two days later the baby became slightly croupy at night. Diphtheria was suspected and a dose of 10,000 units of antitoxin was administered. Cultures taken from the pharynx showed abundant pneumococci and influenza bacilli, but no diphtheria.

The following day the spasm of the glottis was extremely bothersome, and there was an inspiratory crow and an expiratory croupy cough. Ipecac relaxed the spasm for an hour. After violent spasm she became cyanosed. The antitoxin had no effect and after three days the baby was decidedly worse. The patient was very limp and pale. She had just finished a violent paroxysm of coughing. Within twenty minutes there was a recurrence of the attack. There was a marked inspiratory crow on each respiration, and a severe croupy or barking expiration. The crying voice was hoarse. Cyanosis was marked. Gradually the attacks subsided.

On direct inspection of the larynx the mucous membrane was of a dull grayish red color. There were small stippled patches on the ventricular bands which were much swollen. The vocal cords were only partly visible on account of the constant spasm and were red (Plate 1, Figs. 2 and 5). The subglottic region was very edematous so that the 4 mm. bronchoscope was introduced with difficulty. In this locality there were small patches of sticky mucus. The child made no attempt to cough until the trachea and bronchi were freed of the thick gluelike secretion by suction (Plate 2, Fig. 2). Both inspiration and expiration improved. A flat headed noncough-up tube for a 1-year-old child was introduced after the removal of the bronchoscope. The baby's condition gradually improved and after three hours she was comparatively comfortable, though the spasmodic cough, with the tube in place, was troublesome at times.

A few days later the temperature, which had been 103 F., gradually fell to normal and the tracheobronchitis subsided.

Four days later the intubation tube was removed through my direct spatula. The subglottic edema had not subsided, and within thirty minutes reintubation was necessary. Six days later the tube was again removed as the arytenoids had resumed a normal appearance. As the intubation tube was removed the bronchoscope was introduced and secretion aspirated and the condition of the trachea and bronchi observed. On the gradual withdrawal of the bronchoscope the baby was able to breathe through a fairly normal larynx. The baby had a few attacks of glottic spasm and croup but they were not sufficiently severe to require reintubation. After three weeks the baby had recovered.

CASE 7.—A boy, 3½ years of age, was admitted to the Willard Parker Hospital apparently moribund. On admission an immediate direct laryngeal and bronchoscopic examination was made. There was considerable difficulty with respiration; expiration was prolonged and wheezy. The larynx was deep red in color and cyanosed. There was slight subglottic edema. The 5 mm. bronchoscope was introduced to the carina and revealed absolutely dry trachea and bronchi. There was dusky redness of the bronchi and trachea. There was absolutely no sign of a cough reflex.

Three hours later the child became so much worse and the asthmatic expiration became so bothersome that the bronchoscope was again introduced but without result. This time there was a slight amount of yellowish tenacious mucopus exuding from the left superior lobe bronchus (Plate 2, Fig. 6). All the other bronchi remained dry. Suction cultures showed pneumococci; no diphtheria bacilli were found. Twelve hours after admission the child died.

EXPLANATION OF PLATES

PLATE 1

Fig. 1.—Direct laryngeal view in Case 3. The larynx was slightly cyanotic but otherwise normal in appearance. The vocal cords were normal and the ventricular bands slightly swollen. There was marked subglottic edema. This is shown in the picture as two dark grayish masses projecting from the lateral subglottic walls and well below the vocal cords.

Fig. 2.—Direct laryngeal view in Case 6. The larynx is deeply congested. The vocal cords are reddened and the ventricular bands swollen. The subglottic region is beefy red and swollen and there is a thin exudate in stippled patches on the tumescent mucosa. The arytenoid cartilages are slightly swollen. There was a constant glottic spasm which added to the gravity of the case. The subglottic edema is productive in causing the expiratory croupy bark.

Fig. 3.—Direct laryngeal view in Case 1. The aryepiglottic folds and ventricular bands are swollen. There is a stippled appearance of the larynx and many fine white points are seen on the deep red background. There is moderate subglottic edema present. The markedly injected vessels and white stippled points are plainly seen in the trachea.

Fig 4.—Direct laryngeal view in Case 2. The ventricular bands are slightly swollen, causing inspiratory difficulty. Note the marked annular subglottic and tracheal edematous stenosis which made the introduction of the infant bronchoscope difficult. The vocal cords are normal, but partially covered by the swollen ventricular bands.

Fig. 5.—Direct laryngeal view in Case 6. Showing persistent glottic spasm and the cause of the bothersome inspiratory crow. The swollen ventricular bands overlap the cords and cause inspiratory dyspnea.

Fig. 6.—Direct laryngeal view in Case 4. Note the beefy red larynx with enormous tumefaction of the aryepiglottic folds and ventricular bands. Only a small lumen is left for inspiration, for the swollen ventricular bands while in spasm almost close the glottic inlet. On introduction of the bronchoscope and separation of the tumefied masses the subglottic region was found to be equally edematous. This case had both inspiratory crow and expiratory croup.

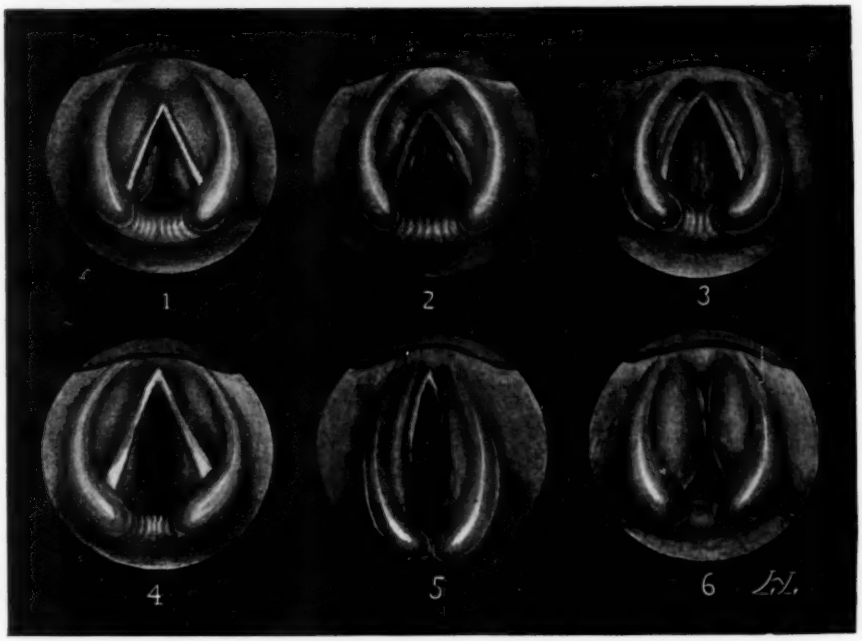

PLATE I

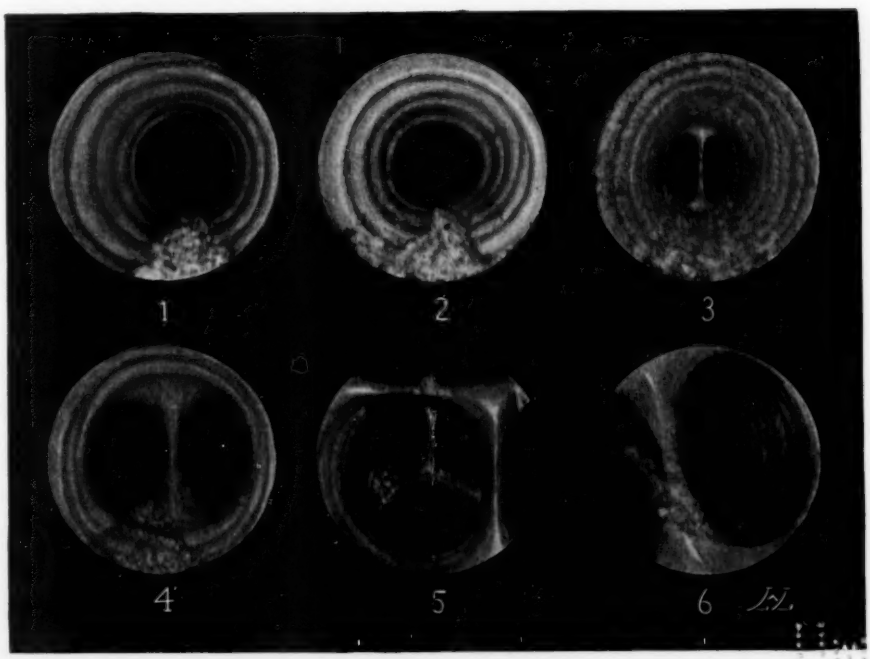

PLATE II

ILLUSTRATING ARTICLE BY HENRY LOWNDES LYNAH, M.D., AMERICAN JOURNAL OF DISEASES OF CHILDREN, OCTOBER, 1919

PLATE 2

Fig. 1.—Tracheal view in Case 3. Note the slightly cyanotic trachea with thick tenacious glue-like secretion of influenza on the posterior wall. The trachea was edematous and there was an asthmatic wheeze even after the secretion had been aspirated.

Fig. 2.—Tracheal view in Case 6, showing tenacious secretion on the posterior wall and hemorrhagic patches on the lateral walls and in between the tracheal rings.

Fig. 3.—Tracheal view looking toward the carina trachialis (Case 1). Note the marked stippling and swelling of the mucous membrane. The white points are pneumococcic stippling on the reddened and blood streaked mucous membrane. There was very little secretion removed by aspiration.

Fig. 4.—Tracheal view at the carina in Case 4. There was no involvement of the right bronchus which was free from secretion. There is a plaque of tenacious mucus partially occluding the left main bronchus. Note the hemorrhagic points surrounding the swollen carina.

Fig. 5.—Bronchoscopic view of the right main bronchus and its branches in Case 3. The stem bronchus and branches are very cyanotic and completely occluded with thick bloody mucopus which obstructs respiration on that side entirely. No air entered the right lung until after the secretion was aspirated bronchoscopically. There was a decided asthmatic wheeze on expiration even after the aspiration of secretion and with the bronchoscope in situ.

Fig. 6.—Bronchoscopic picture of the left main bronchus and left superior lobe branch (Case 7). The child was admitted in a moribund condition. No secretion was found in the bronchi on the first bronchoscopic examination and only a small amount was poured out at the time of the second bronchoscopic examination and then only from the left bronchus. The larynx was not involved. The child was suffering from the bronchopulmonary asthmatic type of influenza and had no cough reflex on the introduction of the bronchoscope. Note the tenacious glue-like mucopus exuding from the left superior lobe orifice.

A HEALTH STUDY OF A BOYS' SCHOOL*

RICHARD M. SMITH, M.D.
BOSTON

The health of schoolchildren has been a matter of interest to physicians and school authorities for many years. This interest has been given an added impetus as a result of the war and the general awakening to the importance of child life. Thus far the care of the health of schoolchildren has been for the most part along conventional lines. The primary effort has been directed toward the detection of physical defects and the listing of them. Notice has been sent to parents telling them of the need for treatment. In some instances treatment has been secured through the follow-up work of school nurses. Recently special school clinics, notably, dental clinics, have been established for correction of defects. This is especially true in communities where hospital facilities are inadequate. This development is a distinct advance over the work of previous years, but there is much still to be wished for in this direction. It has been customary also in some schools to provide lunches. For the last two years there has been unusual interest in malnutrition. This interest has resulted in a considerable measure from the weighing and measuring tests of young children instituted by the Children's Bureau. With the discovery of the prevalence of malnutrition has come a new emphasis on the necessity of proper food. Special classes have been formed for the feeding of underweight children.

In many schools the entire plan, as outlined above, for taking care of the health of the pupils has been in operation, and in nearly all schools some part of it. In only a few schools has there been any study of the health of the children taking into account all factors in the child's life and their relation to one another. The public schools have been more progressive than the private schools in this matter. In fact, many private schools, excellent in other respects, entirely neglect the question of health.

As pediatricians whose chief responsibility has to do with the health of children, there are certain matters concerning which I believe we should take definite action. These matters have to do with the quality of care given at present to the health of schoolchildren and with further needed extension of care. There are certain minimum standards of health supervision to which every schoolchild is entitled.

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*Read at the Annual Meeting of the American Pediatric Society, held at Atlantic City, June 16-18, 1919.

In the first place, we should demand that every schoolchild be given a complete physical examination at least once a year, and means provided for the correction of defects and the treatment of diseases found. At the same time we should see to it that defects or diseases do not develop while the child is in school. Physical examinations should be made by a competent physician and made with the child undressed. I am told that at a recent meeting of a state medical society a well known school physician in a large city argued that it was impossible to undress schoolchildren for examination, and that we must content ourselves with the imperfect results obtained by unsatisfactory methods. I do not believe parents have any objection to an examination, provided they understand why the examination is made. Good medical advice cannot be given without careful examinations.

In the second place, we should insist on proper qualifications for school nurses. School nurses are really health instructors, and they must be selected because of efficiency in that work, not because of any other reason.

In the third place, we should bring about a better understanding and a closer cooperation between the school authorities and the family physician, and the practicing dentist. Often the advice given at school and that given at home are entirely different. The difference usually arises because of a failure of understanding. Unity of action must be secured for the good of the child's health. If minimum health standards for schoolchildren were established and taught, there would be no conflict of advice. This involves the introduction of the teaching of school hygiene in the medical schools — a development which is sure to come in time, and which I believe we should try to secure at once.

In the fourth place, we should continue our recent interest in the question of nutrition. We must recognize in this connection that the study and special care of undernourished children does not include the whole problem. It is not sufficient to weigh and measure all schoolchildren and then confine all our attention to those who prove to be underweight. This method gives no recognition to the equal importance of preventing malnutrition in those who at the present time may be of normal weight. It is to be borne in mind also that our standards for weight and height are by no means positively established. It is quite possible that the true normal may be of wide variation. We must, therefore, see to it that all the children are in good nutritional condition. To this end school lunches of the right kind should be provided for all the children, and some supervision given to the methods in which the lunch is eaten.

In looking toward the future extension of health supervision there are certain additional features to which we must direct our attention.

First.—There must be a general recognition of the importance of health. The recent draft has shown that our present methods of safeguarding health do not produce satisfactory results. The army required healthy men as its requisite for service. Health is just as necessary for efficiency in peace as in war. This principle is fundamental for further progress.

Second.—There must be a realization among school authorities, both private and public, that the health of children is as important as scholarship, and that education must not go at the expense of health. In many individuals this will involve a complete change in point of view with reference to school problems, and in many instances a radical readjustment of the school program.

Third.—There is especial need of securing the cooperation of parents in protecting the health of their children. In general, parents have failed to realize the importance of health in connection with school. I believe this is due to lack of information. They are particularly careless in the planning of activities outside of school hours, such as special lessons and parties. These activities must be planned in connection with the school work, and not be added to that without reference to other matters. The child's life cannot be divided between school and home hours, and each planned independently, if good results are to be obtained. The school master and the parent, and those interested in child welfare, with the advice of the physician, must act together and see that the child's life as a whole is lived so as to protect and not injure his health.

Fourth.—It is important that the pupils themselves receive instruction regarding health. The teaching of health to pupils must be eventually in the hands of the school authorities. This can be done only when the school authorities recognize the importance of health and after they have received from physicians information as to the proper things to teach. This makes it necessary for us to be ready to submit courses of instruction for teaching health to children. Thus far there has been very little thought given to this matter, either by physicians or by anyone else. We must consider it, however, in the immediate future if we wish to have the right things taught.

I have stated my conclusions first, because they seem to me of more value in that position. What I have to say with reference to the particular school is a description of the methods used there to safeguard the health of the children. I have had an opportunity of working with the head of the school in a practical demonstration of the principles which I have described.

Mr. Rivers' Open Air School for Boys in Boston was started in 1915. From the beginning health has been considered equal in importance to education.

There is an advisory medical committee to whom the details of the curriculum are submitted in order that education and health may be considered in relation to each other. The work of the school is conducted entirely in open air classrooms. The food is approved by the medical members of the advisory committee. The children have rest periods each day. This is a definite part of the curriculum, and adapted to the varying needs of the boys of different ages. This is particularly important with the younger boys, who cannot get the full benefit of school without the periods of rest. Each boy's school program is explained in detail to his parents and the importance of planning home time in relation to this program is emphasized. The parents are told that they cannot expect good results in scholarship or health unless they consider the life of the boy as a unit. The school sends the boy to his family physician for advice when the progress in school or the health of the boy is suffering from a physical condition needing correction.

There is a school matron, corresponding to the school nurse in the public school, whose sole duty is to look after the health of the children. She does this under the direct supervision of the head of the school with the advice of the medical committee.

Every pupil on admission to the school brings a medical chart filled out by his parents or by the family physician, a form of which is shown, giving the medical background of the boy. This is considered carefully in planning his school work, his rest periods, his diet and any special supervision which is necessary. Each boy is given a complete physical examination at the opening of the school year. On the basis of this physical examination in conjunction with his medical history his school program is determined, and he is listed in one of several groups, either as "normal" or "for general observation," or as "overtire," i. e., a boy who has to be watched because of the possibility of fatigue, or as "colds," i. e., catches cold easily, etc.

The hours at school are divided into four groups, study, inactive, active and occupation. This is done in the belief that for each child there is a proper balance between his hours of study, activity and inactivity during each day. So far as the school is concerned, this can be carried out accurately. For its complete application it requires the willingness on the part of the parent to cooperate. Although the hours at school represent most of the boy's day, there is time before school and especially after school at night when there may be no supervision. If these out of school hours are given no thought, in connection with

his school routine, his balance of time is disturbed. The number of hours of study for a boy of given age and grade is pretty constant and must remain so if he is to do the required amount of work and advance. The number of hours of inactivity, activity and occupation may be planned according to each boy's requirements. By periods of activity is meant school athletics and recess; by inactivity the rest period and dinner hour.

From a study of the boys in the school over a period of several years it has been determined that the following percentages of time represent about what the average boy of a given age can do without injury to his health and make satisfactory progress in his studies.

TABLE 1.—STANDARD PERCENTAGE OF TIME GIVEN TO STUDY, INACTIVITY AND ACTIVITY

Age	Study	Inactivity	Activity
6 years	4	3	2
7 years	5	2½	2
8 years	5	2	2½
9 years	5	2	2½
10 years	5	2	2½
11 years	5½	2	2½
12 years	5½	1½	2½
13 years	5½	1½	2½
14 years	5½	1½	3
15 years	5½	1½	3

It will be seen as the children grow older, the study hours increase slightly, the periods of rest decrease and the periods of activity increase. Each boy's schedule in percentages is checked against the formula for his age. This method has been of great value in many instances where boys were not doing well. This is not unlike the use which is made of caloric requirements in infants' feeding.

Every boy when he gets to school each morning is checked in on a chart by the matron. At this time inquiry is made as to his health. The matron also makes a superficial estimate of whether the boy looks well or not. If she is in doubt, the matter is pursued further. She sees that his clothes are right for the day. If a boy is not present and the reason is not known, the parents of the child are called on the telephone and the reason determined. The parent is asked, if the cause of absence is illness, where it was contracted. This is done in order that a record may be kept of whether the school was in any way responsible for the illness, i. e., whether it could have been prevented by any modification of the general school program or of that particular boy's program.

There is a general list of boys who need special attention which is posted in every school bungalow in order that the teacher may see and keep in mind the children in his class who require unusual care from

the health point of view. If a boy on any particular day requires additional attention, the matron, at the time of checking in, makes a note of it. A list of these boys is taken to the teachers in charge of their rooms. Each boy when he leaves the school in the afternoon is checked out. At this time the matron sees that he has on enough sweaters to ride home; that he is not too hot or too cold or that his feet are not wet, and that he is physically in good condition. There is also noted in his checking out, if he leaves at any unusual time, the reason for it or any special comment. By this means of checking in and out, I feel confident that a great amount of good results. It makes a particular person responsible for the health of every boy each day. Between the time of checking in and checking out the matron spends her time in the schoolrooms, being sure that the boys who need special attention are receiving it. She sees that they are properly dressed for exercise and in general keeps track of the health program as outlined by the school.

A chart of absences is kept on which is recorded each day all the absences and the reasons in order that it may be visualized to compare with other days. There is recorded on this chart also the daily temperature and the kind of weather. A study of this chart reveals several interesting things. There is a remarkably small amount of contagious disease. This, I believe, is due to the constant effort on the part of the school to exclude any possible sources of infection. Children are kept from school whenever there is known illness of this character at home. The chart shows a variation in respiratory diseases, primary colds, running with modification in the weather. It shows also the small number of cases of illness which can be traced in any way to the school. Table 2 is a summary of the chart.

TABLE 2.—ABSENCES

Total number of boys in the school at present, 115 (slight variation during year)		
Number of school days	142 (Oct. 25, 1918, to June 6, 1919)	
Possible total attendance	16,333 days	
Days lost through colds	859 days	5.2%
Days lost through contagion	197 days	1.2%
Days lost through minor illness	487 days	2.9%
Days lost through quarantine	51 days	0.3%
Days lost through no reason traceable to illness	541 days	3.3%
Days lost through illness traceable to school	94 days	0.5%

There is a table of some of the boys who were below par on entrance, as indicated by being underweight. The progress during the year, the gain in weight, the scholarship record, the number of days absent and the usual reason are also shown. It will be seen that many of the boys made good progress in scholarship but had colds, and that others made good progress despite the fact that they did not make satisfactory gains in weight. A similar table might be shown of boys

who did not make good progress in scholarship or who had many absences due to illness, but who had a normal weight at entrance and made good gain in weight during the year. Some of these boys had a physical handicap accounting for their poor scholarship, which was corrected, and then they did well in school work. These boys would have been neglected in health and in scholarship if the weight only had been considered as an index of health.

TABLE 3.—SCHOOL PROGRESS OF UNDERWEIGHT CHILDREN

Name	Weight Oct. 1918	Underweight for Height	Gain in Weight During Year	Scholarship	Days Absent	Usual Reason
B.	65 lbs. 13 oz.	5 lbs. 3 oz.	2 lbs. 11 oz.	Good	6	Colds
B.	83 lbs. 12 oz.	15 lbs. 4 oz.	9 lbs. 6 oz.	Good	12	Colds
C.	61 lbs. 5 oz.	10 lbs. 11 oz.	3 lbs. 4 oz.	Excellent	7	Colds
G.	53 lbs. 15 oz.	6 lbs. 1 oz.	2 lbs. 3 oz.	Good	12	Colds
K.	56 lbs.	6 lbs.	1 lb. 2 oz.	Excellent	25	Colds and chickenpox
P.	67 lbs. 12 oz.	7 lbs. 4 oz.	2 lbs. 14 oz.	Excellent	6	Colds
P.	65 lbs. 8 oz.	11 lbs. 8 oz.	4 lbs. 4 oz.	Good	6	Minor
McP.	75 lbs. 3 oz.	24 lbs. 13 oz.	8 lbs. 5 oz.	Excellent	2	Minor
P.	88 lbs. 10 oz.	18 lbs. 6 oz.	9 lbs. 10 oz.	Excellent	6	Minor
W.	58 lbs.	4 lbs.	2 lbs. 10 oz.	Excellent	3	Minor
W	58 lbs. 10 oz.	3 lbs. 6 oz.	2 lbs. 3 oz.	Good	5	Chickenpox

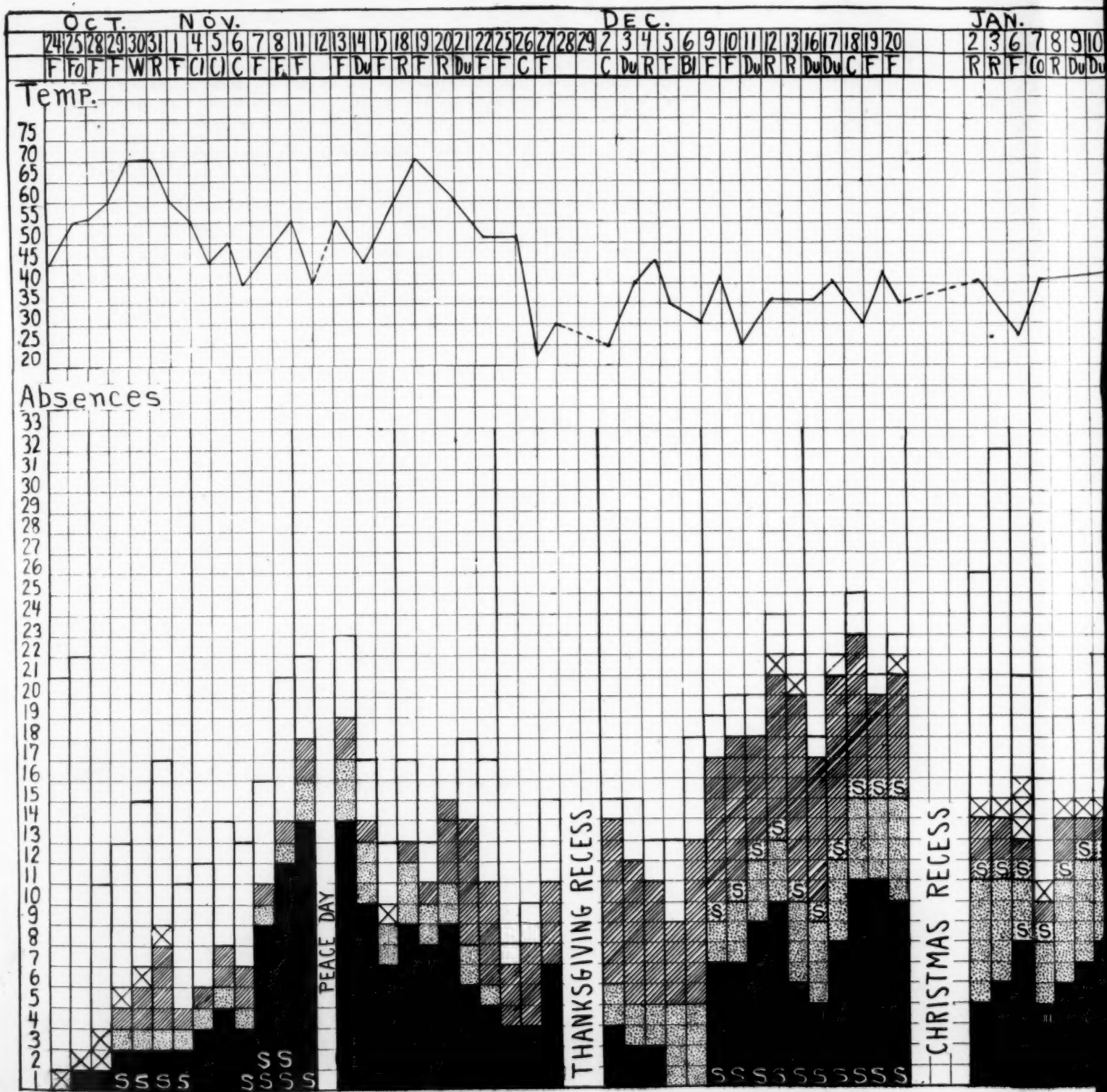
It may be worth while to cite the record of a few boys.

CASE 1.—B., 12 years of age; normal weight for height. Had a history of repeated colds. Physical examination was negative except for a rather "ragged" throat. His progress in school was slow. He was absent often. Colds were cured by medical treatment, and then progress in school was good and he passed from the lower end of the grade to a candidacy for first position. His attendance was normal.

CASE 2.—D., 9 years of age; was underweight for his height (11 per cent.), and in poor general condition. Physical examination was negative except for the undernourishment. He did poor work at school and had many absences. A conference was held with his parents and the physician and it was found that after school at home he overexercised with resultant fatigue and interrupted sleep. His hours out of school were regulated in connection with the school schedule. The result was a marked improvement in school. His health is excellent, although he made only slight gain in weight (1¾ pounds).

CASE 3.—H., 12 years of age, underweight (12 per cent.), with normal past history, except that he was "never robust." Physical examination negative except for the undernourishment. His scholarship was very bad. Absences were frequent. His schedule was arranged with special attention and more rest, and more supervision at meals to insure his taking the proper amount of food. The result was that his scholarship became excellent. His general condition was markedly improved. His weight is within a pound and a half of normal.

CASE 4.—F., 13 years of age; underweight (10 per cent.); gave a history of repeated colds. Physical examination was negative except for chronic infection of the tonsils with enlarged cervical glands. He did poor work at school and was absent repeatedly for tonsillitis. His tonsils were removed. His progress in school became excellent. He had no more absences and gained to within one and a half pounds of normal weight.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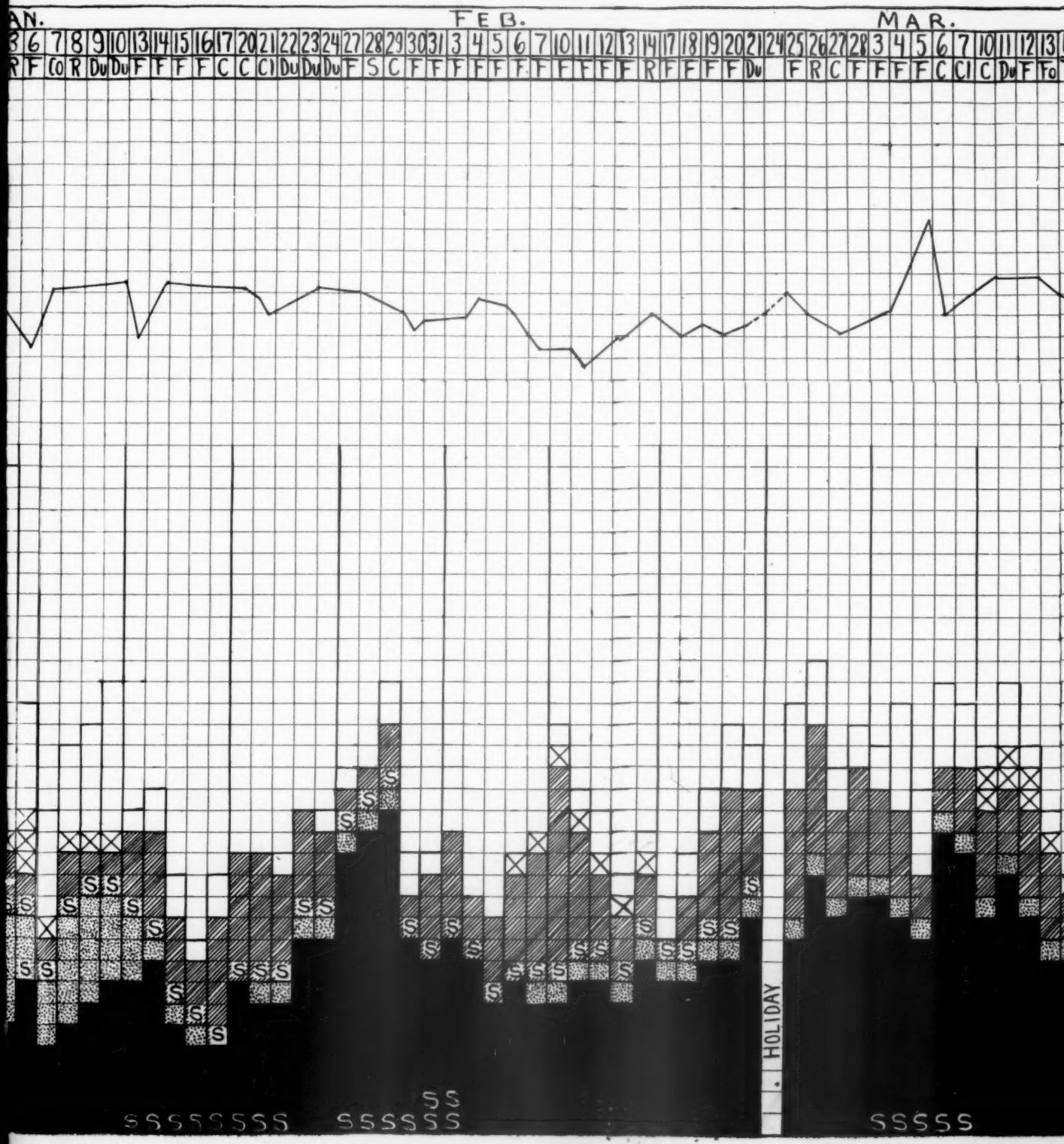
PEACE DAY
THANKSGIVING RECESS
CHRISTMAS RECESS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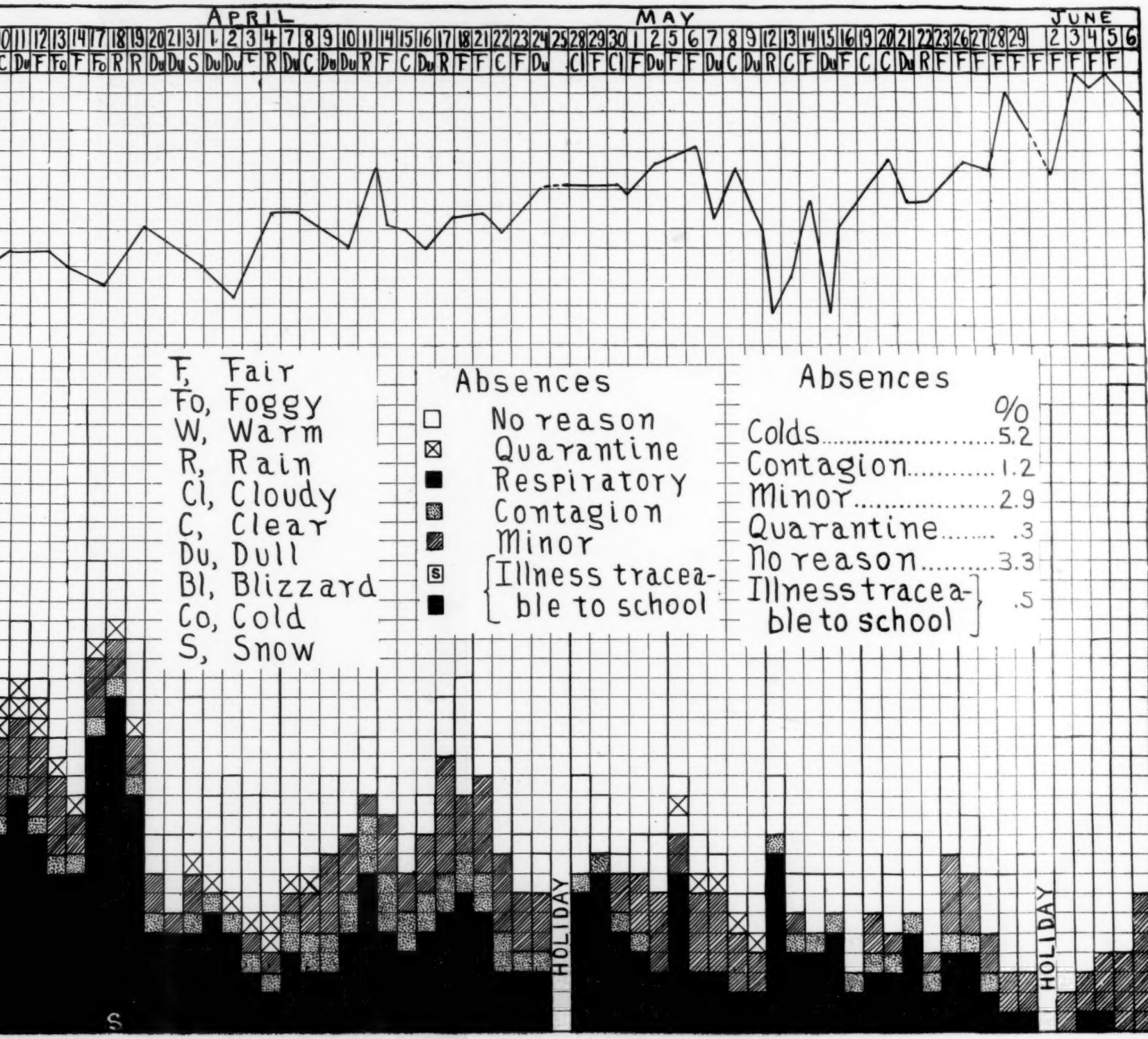
APRIL
MAY
JUNE
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Fo, Foggy
W, Warm
R, Rain
Cl, Cloudy
C, Clear
Du, Dull
Bl, Blizzard
Co, Cold
S, Snow
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No reason
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Minor
Illness traceable to school
Absences
%
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HOLIDAY
HOLIDAY

CASE 5.—P., 12 years of age; underweight (24 per cent.); had a history of repeated absences from school. He never felt well and made very poor progress in the classroom. Physical examination was negative, except for the marked undernourishment. It was quite evident that the difficulty with this boy was malnutrition and that he was constantly overtired. His schedule was made out with this in mind. By Christmas he began to show the effects of the special care. During the second half year he had no absences, and he is now one of the best students in the school. His gain in weight was only fair. He is now 16 per cent. underweight. Special watching by the matron made the change in this boy.

It is difficult to compare results from the health standpoint in this school with those in other schools, because of the lack of uniform standards of estimate. I am convinced, however, that progress in school health supervision must be in general along these lines: first, recognition by school authorities of the importance of health and the necessity of arranging school work with this in mind, and second, cooperation of parents and those interested in children in planning the time out of school in relation to the school program.

329 Beacon Street.

CERTAIN NUTRITIONAL DISORDERS OF CHILDREN ASSOCIATED WITH A PUTREFACTIVE INTESTINAL FLORA*

LANGLEY PORTER, M.D., G. B. MORRIS, M.D.
AND
K. F. MEYER, M.D.
SAN FRANCISCO

It has long been recognized that the intestinal flora of the very young varies, and that the character of the ingested food is one factor in the production of this variation. The description of acidophilic, gram-positive organisms in the stools of breast fed infants by Escherich[1] and Moro[1] are classic, and the same authors, as well as Bahart,[1] Giarre,[1] Concetti[1] and Schikora,[1] with equal clearness have described bacteria to be found in the stools of artificially fed infants. Thus it has been common knowledge for many years that infants fed a diet rich in bovine milk acquire a characteristic stool flora which in contrast to that of the nursling is putrefactive in its activities and which produces chemical substances (indol, phenol, skatol, etc.) which are known to arise from the splitting of protein. Giarre,[1] Schikora,[1] Langstein,[1] Soldin[1] and Bookman[1] all recognize the antagonism between the acidophilic flora produced under human milk and other high sugar feedings and the alkali loving proteolyzers that inhabit the gut of children taking bovine milk or other foods high in animal protein. Finkelstein used the powers of high casein as contained in his albumin milk to check acid diarrheas brought about by an excessive carbohydrate intake; and for years many of the putrid diarrheas have been treated empirically by the use of cereal decoctions, or of maltose or lactose solutions; the rationality of this procedure is explained if we turn to the results of Kendall[2] and of Torrey,[3] who have shown that under an exclusive carbohydrate regimen, putrefactive organisms may disappear from the intestines of experimental animals.

We have long felt that there was clinical reason to suspect an etiologic relationship between protein splitting bacteria and certain

*From the Department of Pediatrics and the George Williams Hooper Foundation for Medical Research, University of California Medical School, San Francisco, Calif.

1. Quoted by Tobler: "Algemeine Pathologische Physiologie der Ernährung und des Stoffwechsels im Kindesalter." Bibliography, pp. 236 to 247.
2. Kendall, A. L.: J. M. Res. **25**:117, 1911.
3. Torrey, J. C.: J. M. Res. **39**:415 (Jan.) 1919.

types of alimentary intoxications that occur during the earlier years of life. The types of alimentary intoxication in which we have been interested and which we have had an opportunity to study, are:

(a) A mild type with constipation, lack of gain, pallor, head sweating and loss of turgor, the so-called balance disturbance of the Finkelstein school.

(b) A fulminant, acute type with sudden onset, profound cumulative toxemia, tremendous shock, alarming loss of weight and too often an early fatal issue with acidosis.

(c) A grave, chronic type with diarrhea which occurs for the most part between the tenth and twentieth months, characterized by frequent stools, anorexia, progressive loss of weight and increasing intoxication. Previous authors have classed these cases as "Cœliac disease, pancreatic insufficiency and chronic alimentary intoxication."

(d) Certain sporadic, acute, putrid diarrheas of young infants.

(e) A type of chronic malnutrition to be found in older children, a type which in its clinical features corresponds to the balance disturbance of infants, but in which the symptoms are modified by the difference in the ages and dietetic habits of the patients.

The most important and puzzling to explain of these clinical pictures is undoubtedly the acute type of alimentary intoxication, with the dramatic suddenness of its onset, its rapid course, and its tragic termination in death with acidosis. It was this type of case that first fixed our interest in the subject.

Many years ago one of us was struck by the resemblance between the symptoms of intoxication as they occur in this disease and in acute intussusception of infants. Two patients lay side by side in the wards of Lane Hospital, one brought in with an intussusception of some days standing, the other a victim of acute alimentary intoxication. Both were robust, well developed children. Both lay in profound shock, pale, with sunken eyes, flaccid, slightly cyanotic about the mouth, semiconscious; once roused both quickly relapsed into stupor. In the case of the child with intoxication, the symptoms of bowel obstruction failed and the respiratory evidences of acidosis were more marked in type and more rapid in onset. In other respects there was a startling resemblance in the clinical aspects of the two cases. The similarity was so striking that it emphasized the idea that a similar toxin might be working in the two cases, so that later, when Whipple reported his views on the cause of death in bowel obstruction with the experiments that led him to incriminate a toxic albumose, it was natural to search for a possible source of toxin formation, and with Torrey's classical research on the damaging effects of a high proteolytic flora in the intestines of patients, victims of typhoid infection, it was

equally natural to initiate an investigation of the rôle putrefactive organisms might play in acute alimentary intoxications.

In San Francisco the acute fulminating types of intoxication are rare, so that the deductions drawn from the few cases on which this part of the study is based can be only inconclusive, although the facts elicited are striking enough to be suggestive, especially when they are taken with the results obtained from the study of a large number of the more frequent subacute types of intoxication.

The opportunities afforded one of us to study the intestinal bacteria of the three earlier cases were inadequate, but in all the change from the normal flora was striking. The stools of all showed putrefactive organisms in great predominance. All three of these patients died, and in each case characteristic necropsy findings were revealed, great increase in fat in the liver, acute degeneration of the kidney with fatty infiltration, and an almost pure proteolytic flora in the lower ileum.

Shortly after the death of these patients the other collaborators became interested in the problem, and they have developed the bacteriologic and experimental facts here presented.

Following these investigations a system of treatment was devised and in nine cases during the last two years there has been but one fatality, and that within the last month. We feel that the failure in this case was due to the fact that the patient came to us late in the course of the intoxication. The plan calls for lavage of the stomach with a 2 per cent. sodium bicarbonate solution, and the washing out of the bowel with from two to six quarts of a 5 per cent. solution of the same salt, two or three times a day. All food is withdrawn, except that the patient is given large quantities of an iced, 7 per cent. solution of lactose with 2 per cent. of the sodium bicarbonate solution by mouth, and this is continued in spite of the vomiting. We find that the patients suffer less if they are supplied with large quantities of fluid, and the vomiting ceases more promptly, especially if the ingested fluid is kept very cold.

The intravenous injection of isotonic solution of glucose we have made a routine, even in those cases in which some sugar appears in the urine. If the respiratory signs of acidosis become alarming, Fischer's solution is injected into a vein in a quantity appropriate to the age and weight of the patient. Undoubtedly, the fact that most of these patients were seen before serious acidosis had set in made the treatment more effective, but none the less they were all alarmingly ill and the improvement in each case began at a time when we were able to show that the intestinal flora had altered in type from putrefactive to fermentative.

The following case is illustrative of this type:

CASE 1.—Baby Lane, a patient of Dr. Herbert Yerington, was seen with him at the Lane Hospital in October, 1918. The child was 3 months of age and weighed 13 pounds. It had been on the breast up to September 27 when the mother developed a fever which was diagnosed as typhoid October 2. On this date the child, which had been nursing in spite of the mother's fever, was removed from the breast. It was isolated and put under typhoid precautions. The examination was negative, except that the history states that the child was pale, with dark circles under the eyes, somewhat lethargic and the musculature was soft and flabby and the patient had a toxic appearance. There was nothing striking about the temperature, pulse or respiration. There were several stools which were liquid and had a foul odor. The Widal test was negative and the stool culture showed no typhoid organisms.

This child was put on 5 ounce feedings of a certified cow's milk mixture of approximately 2-1.75-2.5. From October 2 to 5 the only change was a slight rise in temperature, loss in weight and a fall in the number of white blood cells to 4,000. The stools were still frequent but more solid and very foul. Castor oil was given and the milk reduced and October 7 it was noted that the child's condition was improved. October 8 the condition was about the same, but the child looked more toxic. October 9 protein milk (albumin milk), 20 ounces, water 14 ounces, Dextri-maltose ½ ounce, was given, and the next day the child had twelve liquid stools and eight on the following day with great loss in weight, and depression of the fontanels and anorexia developing.

October 12 the child was about the same and was given a colonic flushing and paregoric and caffein. The formula was changed to a condensed milk in dilution, alternating with a wet nurse breast. October 13 there was no improvement, anorexia was profound, what food was taken was vomited and the stools were very foul. The stool was again searched for typhoid and no colonies were found. However, the examination showed a preponderance of proteus and some streptococci. The same day at 11 p. m. one of us saw him for the first time. The child was very toxic, restless, crying occasionally, throwing the head from side to side, and then relapsing into lethargy. The fontanel was retracted, the lips were dry, there was an extreme pallor, the temperature was 100 F., the respiration was irregular and the child had the appearance of impending death.

It is interesting to note the onset and rapid culmination of these more profound symptoms after the use of protein milk. The child was immediately ordered a massive lavage of the bowel with alkaline solution, a Murphy drip of 5 per cent. glucose, the subcutaneous injection of physiologic solution of sodium chlorid and by mouth a 10 per cent. solution of Dextri-maltose in water, 3 ounces every two and a half hours, and frequent small doses of water by mouth from a medicine dropper. It is noted on the history that October 14 the general condition was greatly improved, the toxemia diminished and that the bowel movements had become fewer in number and less foul. Small doses of brandy and paregoric which had been given before were continued, alternating every four hours. A small amount of breast milk was added to the maltose mixture, but was withdrawn the next day.

October 20 the toxemia had entirely disappeared, the child's stools were two in number, soft, yellow and without mucus. October 24 the child was able to tolerate a mixture containing one-third milk, but with a very high percentage of carbohydrate. December 2, when the child was returned to a normal formula, the picture was that of a healthy, happy baby gaining an ounce a day. Coincident with this reestablishment of well being, there was an entire displacement of the putrefactive organisms present during the height of the illness. Such a case represents the less grave form of acute alimentary intoxication. Cases with fulminant onset which run a more rapid course, depart from this picture only in degree.

However, there is still another clinical entity which should be classed as an alimentary toxemia, in which the progress of similar symptoms is slow, and the onset insidious. In such patients the development of a profound toxemia runs through a period of weeks, sometimes months, until it finally merges into a state of chronic malnutrition, or ends in death from starvation. In cases of this type the most striking symptoms are anorexia, occasional vomiting, and a mild diarrhea in which the stools are pale, usually foul, sometimes with a curious mousy odor, rarely odorless. Very often a large quantity of liquid is evacuated with a smaller mass of fecal material imperfectly mixed with mucus; in the severest cases the mucus may contain more or less blood and pus, and in the later stages of the attack undigested food appears in large quantity; at first fat is not wholly split; as time goes on, unaltered fat appears in greater quantities, and later unhydrolized starch and meat fiber also are to be found. Naturally, with such a failure of digestion, weight is lost rapidly while earthly pallor, sweating and the other facial indexes of intoxication appear. A few of these patients develop an intensely itching erythema of the skin, and together with this a curious lethargy with what might be called a pseudocatatonia. These children assume the most extraordinary postures and maintain them for hours at a time. Necropsy on such a patient revealed no abnormality other than moderately large mesenteric lymph glands, fatty liver, and a preponderance of putrefactive flora in the ileum.

An example of this more usual type of chronic alimentary intoxication is:

CASE 2.—G. S., 17 months of age, had an uneventful history until the tenth month when he was weaned from the breast and given a feeding of equal parts of top milk (about 10 per cent. fat), and water with two teaspoonfuls of lactose to each ounce feeding, which was given every three hours. He did well until March 1, 1919, when he developed vomiting and diarrhea. Six to eight very offensive stools were passed daily together with much gas. Reduction and dilution of milk and the prescribing of castor oil produced no amelioration. The child was depressed, irritable and "very sick."

He continued to fail while the symptoms increased in intensity until April 8 when one of us saw him for the first time. He was then very toxic, he vomited ingesta, even water. He alternated between restlessness and lethargy. His breath bore a distinct odor of acetone and his temperature ranged around 103 F. He showed an extreme loss of fluid and a lack of turgor that was striking. The eyes were sunken and ringed with dark circles, otherwise no pathologic changes could be found by the methods of physical examination. There was a marked degree of muscular hypotonia, the child assumed the most unusual attitudes and maintained them for hours—a condition that might be denominated pseudo-catatonia.

Laboratory findings were negative except for a high degree of acidity in the urine, no acetone, and a predominantly putrefactive flora in the stool.

For two days the child's intake was limited to fluid and lactose, 2½ ounces a day given in the form of "high calory" orangeade. Three hundred c.c.

physiologic solution of sodium chlorid was given by the intraperitoneal route on each of three occasions during this time. Six quarts enteroclyses of 1 per cent. of aluminum acetate were used daily, and on alternate days 2 drams of castor oil with 5 drops of turpentine were given. The diet was made more liberal, other carbohydrates were added, and an egg yolk was given once daily. No milk. The patient went on for about three weeks; the afternoon temperature rose to 101 and 102 F. and an indurative erythema appeared on the skin. This itched intensely but responded to the application of compresses of 1 per cent. phenol in glycerin and water.

After three weeks of this treatment, during which the toxemia subsided, the vomiting stopped and the bowels ceased to evacuate abnormally; then came a sudden attack of diarrhea. The stools during this attack showed a high putrefactive quotient. Castor oil and sodium bicarbonate lavages checked the looseness of the bowels. After one day's starvation, one-half ounce more lactose daily was added to the diet. In a few days, cereals, Italian pastes, fruit sauces and jellies were added. Ten days later a stool examination showed a very slightly putrefactive flora. The clinical signs were improving daily and the child left the hospital in good condition—a normal, convalescing baby.

In childhood there are other more numerous cases which present various clinical aspects; these may be classed as subacute or chronic alimentary intoxication. In certain of them the clinical picture is dominated by a diarrhea whose chief distinction is its exceedingly fetid odor. Mild, evanescent and recurrent forms are not infrequently found among infants during the second and third half years of their life. Such cases are much more numerous than is generally supposed, and the reputation of many proprietary foods as valuable in diarrhea depends on the fact that high carbohydrate feeding, especially dextrin feeding, is effective in checking them. The butter-mehl soup of Ochenius is also effective for the same reason.

Clinicians have always found such cases, especially the severer ones, exceedingly difficult to treat, but since we have been able to submit the stools to the tests for putrefactive bacteria, and have found that in all of them some one or other of the putrefactors occurs in great predominance, treatment has become more effective. In the very severe cases practically no other than putrefactive organisms inhabit the gut. Acting on the information derived, we have instituted high carbohydrate, mainly lactose, feeding, and at the same time have withheld or limited the animal protein intake. And almost without exception we have had relatively rapid recovery with no recurrence. The following case is illustrative.

Case 3.—V. S., 14 months of age, referred by Dr. Shade, Oakland, Calif., was breast fed till 11 months of age. Prior history was uneventful. The child had not gained since removal from the breast three months before. She was on a usual diet for her age, including cereals, broth, soup, a little vegetable puree and much milk. Blood and urine were not strikingly abnormal. The stools were typically proteolytic to cultural tests. She had an intermittent fever. The breath bore an acetone odor. There was persistent loss of weight,

increasing pallor and anemia, lack of turgor, and lethargy alternating with periods of irritability.

She was given a high carbohydrate diet, but as this was one of our earlier patients and we had not yet come to a full realization of the part milk protein plays in these cases, she was allowed 20 ounces of milk in her formula. Gray powders and Bulgarian cultures were also given. Two weeks of treatment along these lines followed. The vomiting became more persistent, the stools increased to from twelve to fifteen daily; they were very foul and great quantities of fluid were passed with them. Anorexia became positive and weight was being lost in an alarming manner so that gavage was resorted to of necessity. At this time all animal protein was withdrawn, a 2 per cent. sodium bicarbonate solution with brandy by mouth was the only ingesta allowed for twelve hours. Then for another twenty-four hours only this with the addition of iced, 5 per cent. lactose solution was given. Sodium bicarbonate enemas were also given.

The child began to mend slowly. The lactose was now given in the form of orangeade, 3 ounces of the sugar each twenty-four hours. Cereal decoctions, dextrins and other carbohydrates were added. Little by little the symptoms disappeared and at the same time the stool flora showed an increasing movement toward fermentative types. It was more than sixty days before this child was really free from the toxic symptoms and about four months before it resumed a satisfactory state of nutrition. Since then, however, the child has done remarkably well and is fat, healthy and happy.

Still another group of malnutrition cases with symptoms of mild intoxication occurs in young infants exclusively bottle fed, who are constipated, fail to gain weight, are pale and sweat about the head, and exhibit a loss of turgor. These children would be classed by Finkelstein as babies suffering from balance disturbance. Their stools always contain a much higher proportion of protein splitters than do the evacuations of more perfectly nourished babies. Such infants show rapid improvement with diminution of symptoms when they are put on a higher carbohydrate feeding, provided that the sugar in the food is increased gradually.[4] The success of malt soup feeding is most striking with this type of malnutrition, and the result depends on the replacement of a proteolytic by a predominant saccharolytic intestinal flora, and we have many case records to illustrate the rapid improvement of such patients when they are given an appropriate diet.

Among older children there are often encountered individuals whose symptoms suggest the activities of a toxic agent. A proportion of the children classed as neurotic and delicate present this aspect. On close examination their symptoms coincide in a striking manner with those of younger children who are suffering from the chronic types of alimentary intoxication just related. These patients are usually of the school age and they are brought to the physician because of lack of energy, sometimes because of irritability and naughtiness, at other times for lassitude and malaise. Irritability and bad behavior are often indexes of fatigue rather than of excessive spirits. On

4. Porter, L., and Dunn, C. H.: Am. J. Dis. Child. **10**:77 (Aug.) 1915.

physical examination the children are usually underweight, and there may have been no gain for months, or even for years. Their appetites are capricious, most often they suffer from constipation often alternating with diarrhea. Their color is invariably poor, although the hemoglobin percentage is not always low; there seems to be a toxic vasomotor disturbance which produces this pallor; but the most striking thing about the appearance of such children is the sunken, dark circled eye. In our experience these little ones have always shown a predominant proteolytic flora, and in every case they have responded to a regimen which included rest in bed with a high carbohydrate feeding. In all instances such patients have remained well so long as the flora in the intestines showed a reasonable proportion of aciduric organisms, and they have relapsed whenever they were returned to a high milk feeding. Such an instance is:

Case 4.—J. A., first came under observation with a cystitis when two years of age. She was not seen again until she was 4 years of age when she was brought in because of an intermittent fever. The child has always been constipated and has slept restlessly and failed to gain in weight. Physical examination revealed a child normal except for dark circles about the eyes, a tired look and small, infected tonsils. Stool examination showed a flora predominantly putrefactive.

The child was put to bed and she was given gray powder, ½ grain, three times a day, and she was put on a very high calory diet, consisting exclusively of starches and sugars. This treatment was followed for three weeks without any change in constipation, but the child's physical condition was remarkably improved, she slept better, but no gain in weight was made. After five weeks, sweets, vegetables and fats were added. After a further three weeks the stools showed only a mild proteolysis but the child's constipation was not any better. Acidophilus cultures were given and the constipation began to improve and the stool was reported to be completely fermentative. After another six weeks the girl was put on a diet in which carbohydrates preponderated, and the acidophilus cultures were continued. The constipation had entirely disappeared and there has been no recurrence and her general health is perfect. The child has steadily gained weight.

One other consideration may be discussed. Torrey's[5] original paper was written to demonstrate the evil effect of the high putrefactive flora as a complication in typhoid fever, and from his plea came the utilization of high carbohydrate feeding in that disease. Undoubtedly, in many other diseases, especially during infancy and childhood, the handicap of a putrefactive intestinal flora tends to complicate the clinical picture and to make the condition graver. This is especially true in cases of sepsis in infancy, and undoubtedly in all the acute infections, especially in certain cases of pneumonia and in the bacillary dysenteries of infants, great advantage will accrue from a study of the flora and a substitution of high carbohydrate feeding in place of the excessive quantity of milk that is often fed to these patients. A

5. Torrey, J. C.: J. Infect. Dis. **16**:72 (Jan.) 1915.

case of sepsis so complicated which undoubtedly would have proved fatal without a recognition of these truths was the following case:

CASE 5.—E. G., was referred by Dr. O. Stansbury, Chico, Calif. The family and past history was entirely unimportant up to the onset of the present disturbance. The complaint was that five weeks before she was first seen by us the baby had had what was termed a severe bronchitis with a temperature of 102 F. She was very restless, deeply lethargic and she was continually bathed in perspiration. Her pallor was alarming and she cried as if in pain. She was slightly constipated. There was no diarrhea at any time. The cough gradually improved and the baby became somewhat better. Ten days previous to the day on which she was admitted to the hospital the child had refused to nurse and she then had a slight fever. She again became decidedly lethargic and developed at this time a high grade of photophobia, due to a conjunctivitis. Her turgor was poor because of the great loss of weight evident.

On physical examination we found a rather restless child moaning in her sleep, rolling her head and keeping her eyes continually closed. The conjunctivae were very much reddened. The central nervous system was normal. The eyegrounds were perfectly normal. The temperature was continuously 101 F. in the afternoon. The blood count showed a hemoglobin percentage of 85, with normal white cell count and differential ratio. The urine showed a slight trace of albumin. The stool flora was predominantly putrefactive. The von Pirquet was negative. Streptococci were found in the throat culture. The conjunctivae were not cultured nor was the blood, but clinically this was a typical picture of a streptococcic infection of the blood stream with localization in the upper respiratory tract and the conjunctivae. The case was complicated, however, by the presence of the highly putrefactive flora which we found in the stools.

The child was put on a diet of 7 per cent. lactose solution, alternated with 1.5 per cent. sodium bicarbonate solution, 3 ounces every two hours. This feeding was continued for two days, then 3 per cent. cereal water was added to 7 per cent. lactose, and 0.4 per cent. of protein was allowed. Carbohydrates were given in quantity, fats in moderation, while the protein in the mixture was increased slowly.

The symptoms of the self-limited blood stream infection passed away in about two weeks and the mother was allowed to leave the hospital. She tried to increase the milk in the food to an amount appropriate for a healthy child of this age. Immediately there was a relapse and a return of the symptoms of intoxication. The milk was promptly withdrawn, lactose cereal decoctions, fruit juices and dextrins were given. After one month the turgor was good and the child had regained much of its weight. Its stools were normal, it had lost all its lethargy, had become playful and happy, and at the same time an examination of the stool showed a return to a balanced flora.

While we have been studying the clinical aspects of the various alimentary intoxications for some time, the more intensive bacterial studies are the work of the past year and one half. A detailed account of the bacteriologic methods worked out for the investigation of the problem will appear elsewhere.[6] In this communication we can do no more than indicate the principles on which we have proceeded, and describe a technic which we believe to be accurate and at the same time simple enough for clinical application.

No reference will be made here to the qualitative side of the work which consumed many months and from the results of which we

6. J. Inf. Dis. (in press).

Abbreviated Form of Stool Analysis

Culture Medium	Normal Stool			Putrefactive Stool			Fermentative Stool		
	24 Hrs.	48 Hrs.	72 Hrs.	24 Hrs.	48 Hrs.	72 Hrs.	24 Hrs.	48 Hrs.	72 Hrs.
Gelatin tube	Liquefaction +	Liquefaction +	Liquefaction ++	Liquefaction ++	Liquefaction +++	Liquefaction +++	Liquefaction —	Liquefaction —	Liquefaction +
Loeffler's serum slant	Digestion and discoloration +	Digestion and discoloration +	Digestion and discoloration ++	Digestion and discoloration +	Digestion and discoloration +++	Digestion and discoloration ++++	Digestion and discoloration —	Digestion and discoloration —	Digestion and discoloration +
Cresol-purple milk tube	Acid reaction Gas + Whey expression + Clot soft	Peptonization +	Peptonization +	Acid reaction Gas ± Whey expression + Clot soft	Peptonization ++	Peptonization ++	Acid reaction Gas ++ Whey expression +++ Clot firm	Peptonization —	Peptonization —
1% lactose peptone bile	Gas production 15 to 30 per cent. Smear of sediment: B. coli, gram-positive cocci; few gram-positive rods			Gas production 10 to 25 per cent. Smear of sediment: B. coli, gram-positive cocci; few gram-positive rods			Gas production 10 to 50 per cent. Smear of sediment: B. coli, few aciduric		
Endo plate	Reaction, acid Growth ++ Odor, acid Type of colonies, varied			Reaction, alkaline Growth +++ Odor, pungent and offensive Type of colonies, numerous			Reaction, acid Growth + Odor, strongly acid Type of colonies, few		

were able to take the few qualitative tests that have been combined to form a clinical diagnostic technic. For this purpose it seems four tubes and an Endo plate are amply sufficient. The tubes are two solid ones—one a Loeffler's serum, the other a gelatin stab—and two fluid tubes, the first a 1 per cent. lactose peptone bile, the second a cresol purple milk. The most striking change happens in the latter when such a tube is infected from a predominantly fermentative stool; there is great retraction of the clot, and a maximum expression of whey. After planting from the putrefactive stool the resulting clot is soft, there are few or no gas bubbles, and a trifling amount of whey is forced out; frequently there is stormy fermentation. Seedings from normal stools give appearances in the tube that more nearly approach those of the putrefactive tube than of the fermentative, although they are in themselves distinctive.

The lactose peptone bile gives most aid when the gram smears of the sediment are studied. Here we find the representation of the viable flora of the stool. In proteolytic stools the picture is dominated by spore bearers, gram-positive cocci and *B. coli*. When the gas in this tube exceeds 60 per cent. we can be sure that *B. welchii* predominates in the stool.

When smeared from a putrefactive stool, Loeffler's serum tubes after seventy-two hours show marked digestion, which has been recognizable from the twenty-fourth hour. They present a characteristic dark brown discoloration. The odor is also pathognomonic. It is strong and very offensive. Fermentators growing on this medium cause no digestion, produce no odor and thrive poorly.

Gelatin stabs from fermentative stools have remained unchanged, while those made from putrefactive evacuations are completely liquefied. Liquefaction begins during the first day and is complete by seventy-two hours. The Endo plates give such striking contrasts when streaked from proteolytic and saccharolytic stools that we feel that they will be a great aid in routine diagnosis. The putrefactors produce a strong, characteristic mousy, musty odor from an abundant growth, which causes an alkaline reaction to the medium, thus leaving it unchanged in color. While, on the other hand, fermentative organisms on this medium grow scantily and give a strong odor of acetic acid, and because of this acidity produced a color change from the normal pink to a deep red.

These simple tests, which can be done in any office laboratory, enable us to verify our clinical suspicions quickly and with certainty.

We have been enlightened and encouraged by the experimental data which Torrey[7] has recently supplied. Hhis studies amplify and

7. Torrey, J. C.: J. M. Res. **39**:415 (Jan.) 1919.

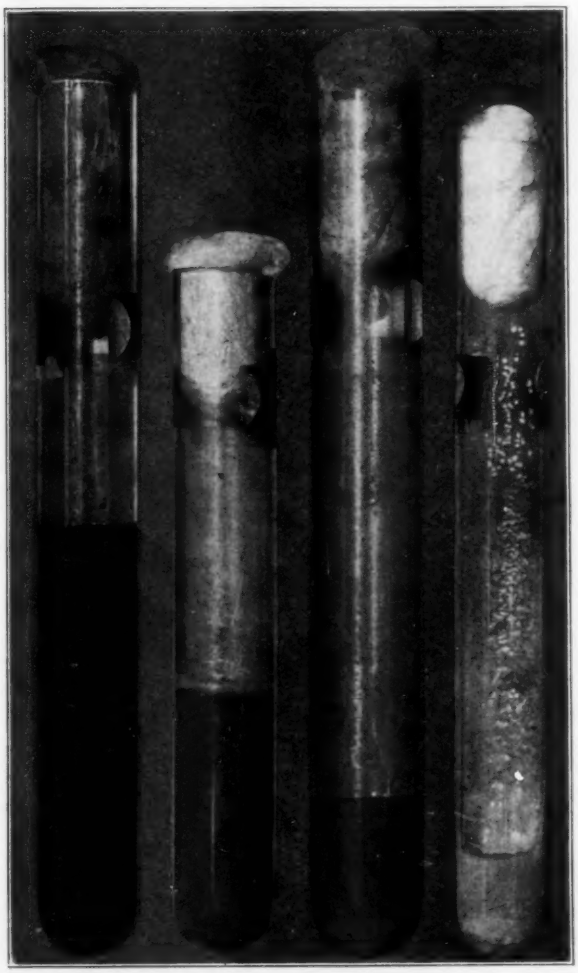
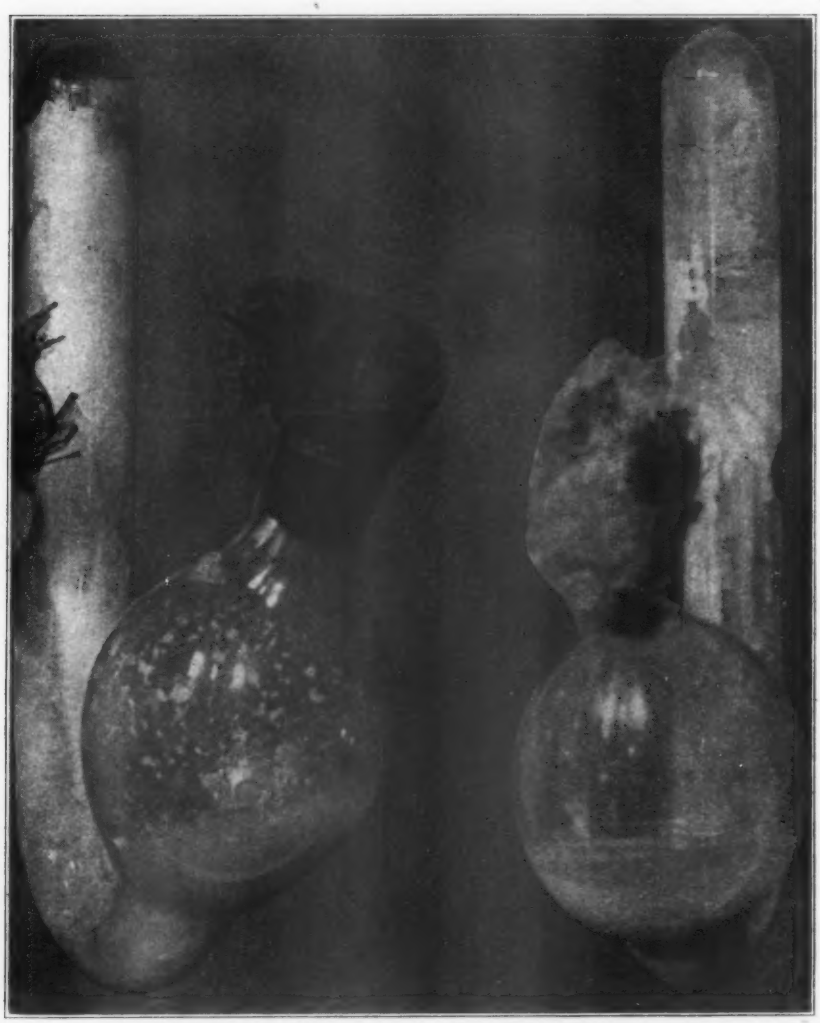
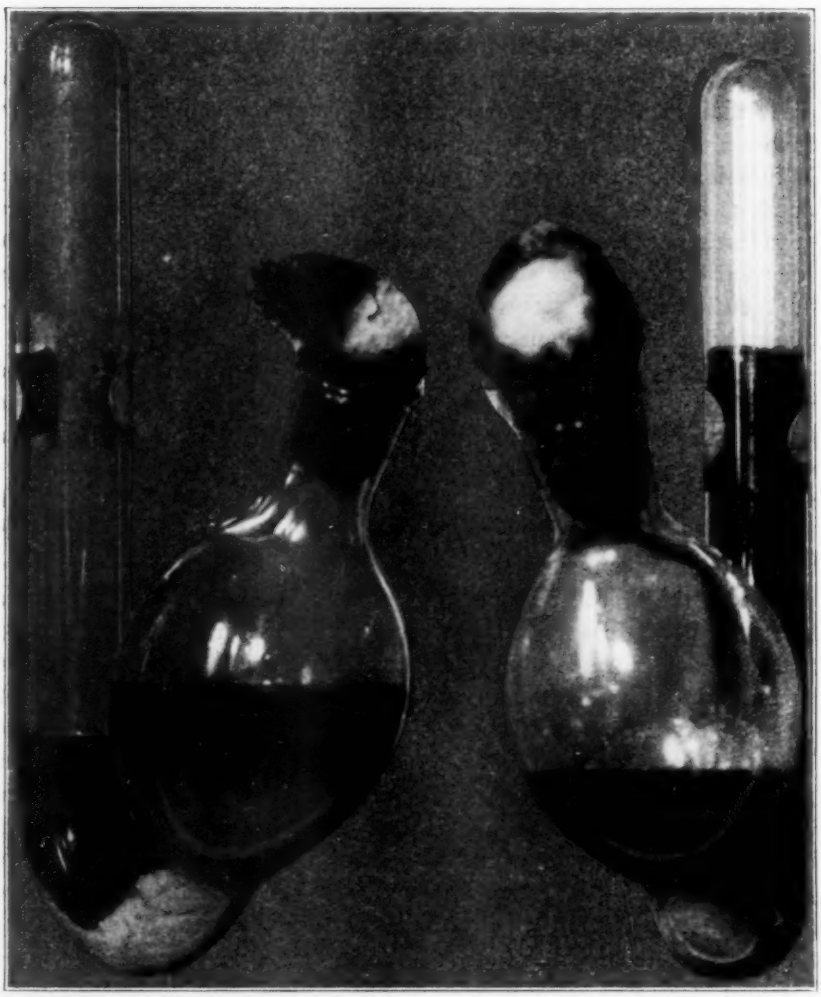


Fig. 1.—A1: Gelatin tube inoculated with putrefactive stool specimen. Complete liquefaction and uniform turbidity. A2: Gelatin tube inoculated with a fermentative stool specimen. No liquefaction and surface growth. B1: Loeffler's serum tube, complete proteolysis of serum produced by bacteria of a putrefactive stool. B2: Loeffler's serum tube, absence of proteolysis—fermentative stool.



A B

Fig. 2.—A: Cresol purple milk—result of a putrefactive stool specimen. B: Cresol purple milk—result of a fermentative stool specimen. Note the expression of whey.



A B

Fig. 3.—A: Lactose peptone bile—gas production when inoculated with fermentative stool specimen. B: Lactose peptone bile—gas production when inoculated with a putrefactive stool specimen.

reinforce the conclusions that we have been able to draw from our own experiences. His data, fully presented, enable him to state as a fact that in dogs the intestinal flora responds promptly and with uniformity to dietetic changes. In his dogs he was able to produce an acidophilic flora at will by feeding lactose and dextrins. He states that one would expect to find a more clearly marked response to food stimuli in the flora of stools taken from the comparatively short canine colon than in those evacuated from the longer human intestine which favors stasis. Torrey further points out the fact that while *B. welchii* is functionally fermentative, it is found always associated with large numbers of putrefactors—a finding which, perhaps, explains why the apparently paradoxic method that calls for two days pure protein diet followed by high sugar feedings is successful in eliminating an excess of Welchii from the stools as well as in removing the toxemias produced by the concomitant putrefactors.

After a study of the facts so far derived from their observations, we feel that we have been able to show (1) that children whose diet is well balanced and whose nutrition is normal, have an intestinal flora consisting of fermentative and putrefactive types without preponderance of either; (2) that children fed on large quantities of cow's milk have a more complex flora made up of various types, most of which are facultative putrefactors; (3) that in children who suffer from certain of the types of alimentary intoxication with malnutrition, the intestinal flora departs in a uniform manner from the normal, and that this departure is always characterized by the establishment of bacterial types predominantly putrefactive; (4) that the return of these children to normal health is coincident with a regression of the intestinal flora toward predominantly fermentative types and a later swing to balance between the two types; (5) that such changes in the intestinal flora can be brought about in the intestine of the human infant by withdrawing animal protein and persistently feeding large amounts of lactose (from 2 to 4 ounces daily), and other carbohydrates; that the period which may be necessary to produce this variation is from ten to forty days. While feeding acidophilus cultures has in a few cases aided a more rapid establishment of aciduric flora in the baby's intestine, this influence was not very great; (6) that the progressive cessation of the symptoms of intoxication and a return of toxemic patients to nutritional health coincides with the recognizable dominance of a fermentative flora; (7) that lactose and dextrins are the carbohydrates most effective in encouraging the rapid establishment of a fermentative flora in the intestines of infants and children.

240 Stockton Street—Second and Parnassus Avenues.

STUDY OF THE STOOLS IN CHILDREN'S INSTITUTIONS SHOWING THE INCIDENCE OF INTESTINAL PARASITIC INFECTIONS

L. R. DEBUYS, B.S., M.D.
Professor of Pediatrics, School of Medicine, Tulane University
NEW ORLEANS, LA.
AND
H. L. DWYER, M.D.
KANSAS CITY, MO.

This study consisted in the examination of the stools of 595 individuals in seven institutions. The ages of the inmates varied from three months to eighteen years and included institutions in which there were both males and females.

The method of examination was as follows: The stools were secured at the various institutions and placed in pill boxes, and each box was labeled with the name of the individual from whom it was obtained. The stool was thoroughly macerated in distilled water and filtered through several thicknesses of gauze. The filtrate was then placed in a centrifuge with a velocity of 1,500 revolutions per minute for from ten to twelve seconds, after which the supernatant fluid was discarded, the residue again thoroughly mixed with distilled water and again centrifuged for a like period. If the fluid after the second centrifuging continued cloudy the residue was again washed and centrifuged a third time. Then the water was poured off and the residue spread on a slide. The examination was made with a minimum of light. This precaution was taken so as not to miss *Hymenolepis nana*, which is very translucent. The preparation was made thin enough for newspaper print to be discernible through it.

The specimen was then placed under the microscope and examined with the lower power to find the ova and then with the higher power for closer study and differentiation.

The time required for the examination of a specimen varies considerably depending on the severity of the infection and the character of the stool. Stools from purges are more difficult to examine. If watery, the preparation is cloudy and if there is much mucus it probably interferes with the ova being thrown to the bottom of the test tube. In this series no stool was considered negative until about one-half hour's search failed to reveal anything. Cercomonas and trichomonas were found in about fifteen instances in the stools examined.

Observations were made on the test tubes in which heavily infected specimens were contained to determine the possibility of ova being carried over in subsequent examinations and increasing the positive findings. It was shown, however, that after being washed and boiled the findings were negative on repeated examinations. This dispelled the possibility of the ova adhering to the sides of the tubes being hard to remove. No tubes were used twice on the same day.

In making the examinations of blood the stool findings were not kept in mind, the counting being done independently.

The stools were secured from seven institutions, which included four orphan asylums, one infant asylum and two waifs' homes. The number of inmates in the institutions were, respectively, 95, 125, 75, 67, 110, 51 and 72; a total of 595.

Of the 595 individuals whose stools were examined, 317, or 53.27 per cent., were infected. Of the 595, 211, or 35.47 per cent., had single infections; eighty-two, or 13.78 per cent., had double infections; twenty-one, or 3.53 per cent., had triple infections, and three, or 0.5 per cent., had quadruple infections.

Two hundred and nine, or 35.1 per cent., of these individuals were infected with *Trichuris trichiura;* eighty-six, or 14.62 per cent., were infected with *Ascaris lumbricoides;* forty-one, or 6.89 per cent., with *Oxyuris vermicularis;* forty, or 6.72 per cent., with *Strongyloides stercoralis;* eighteen, or 3 per cent., with *Necator americanus;* fifty-five, or 9.25 per cent., with *Hymenolepis nana,* and one, or 0.17 per cent., with *Hymenolepis diminuta.*

Of the 317 instances of infections, 211, or 66.56 per cent., were single; eighty-two, or 25.87 per cent., were double; twenty-one, or 6.62 per cent., were triple, and three, or 0.95 per cent., were quadruple.

As to the type of infection in the 317 instances: *Trichuris trichiura* was offender in 209, or 65.9 per cent.; *Ascaris lumbricoides* in eighty-six, or 27.13 per cent.; *Oxyuris vermicularis* in forty-one, or 12.93 per cent.; *Strongyloides stercoralis* in forty, or 12.62 per cent.; *Necator americanus* in eighteen, or 5.68 per cent.; *Hymenolepis nana* in fifty-five, or 17.35 per cent., and *Hymenolepis diminuta* in one, or 0.32 per cent.

THE INSTITUTIONS

The orphan asylum designated in the tables as No. 1 is an institution in which the most complete data could be obtained. Ninety-five individuals were examined, forty-six of whom were males and forty-nine females. Seventeen showed infections. Their ages ranged from 1½ years to 18 years. The previous environment of the inmates was usually known. In seventy-five instances it was unsanitary; and in six sanitary; in fourteen instances the previous environment was unknown.

The duration of the residence in this institution of those studied ranged from a short period up to twelve years — in the infected cases from six months to ten years.

In the seventeen infected cases in this institution, fifteen were of those whose previous environments were unsanitary, one sanitary and one unknown. The parasitic infection of the seventeen in the group were *Trichuris trichiura* in ten, or 58.82 per cent., of the instances; *Oxyuris vermicularis* in six, or 35.29 per cent.; *Hymenolepis nana* in two, or 11.76 per cent., and *Ascaris lumbricoides* in one, or 5.88 per cent., of the instances. The double infections were one each of *Trichuris trichiura* and *Ascaris lumbricoides,* and *Trichuris trichiura* and *Hymenolepis nana.*

There were no animals in the institution. Raw washed fruit and vegetables were eaten. A dietitian is in charge of the institution. There were among the inmates no crawlers, finger suckers or dirt eaters. The stools had been examined once in the past six years. A systematic medical examination is made about twice annually.

In the orphan asylum designated in the tables as No. 2, 125 individuals were examined, 101, or 80.08 per cent., of whom were infected. There were forty-three males and eighty-two females. Of those infected, forty-three were males and fifty-eight females. It is interesting to note that all the boys showed infections. The ages of the inmates ranged from two to seventeen years. The duration of residence in the institution was unknown. The infections in the 101 cases were as follows: *Trichuris trichiura,* seventy-three, or 72.29 per cent.; *Ascaris lumbricoides,* twenty-six, or 25.34 per cent.; *Oxyuris vermicularis,* nine or 8.91 per cent.; *Stronglyloides stercoralis,* twenty-one, or 20.89 per cent.; *Necator americanus,* eight, or 7.92 per cent., and *Hymenolepsis nana,* fourteen, or 13.86 per cent. The multiple infections were as follows: *Trichuris trichiura* with *Strongyloides stercoralis,* eight times; *Trichuris trichiura* with *Ascaris lumbricoides,* six times; *Trichuris trichiura* with *Hymenolepis nana,* five times; *Trichuris trichiura* with *Ascaris lumbricoides* and *Strongyloides stercoralis,* four times; *Trichuris trichiura* with *Necator americanus,* three times; *Trichuris trichiura* with *Necator americanus,* three times; *Trichuris trichiura* with *Oxyuris vermicularis,* three times, and one time each of the following: *Ascaris lumbricoides* with *Hymenolepis nana; Oxyuris vermicularis* with *Strongyloides stercoralis; Oxyuris vermicularis* with *Hymenolepis nana; Strongyloides stercoralis* with *Necator americanus; Strongyloides stercoralis* with *Hymenolepis nana; Trichuris trichiura, Ascaris lumbricoides* with *Necator americanus; Trichuris trichiura, Necator americanus* with *Hymenolepis nana; Oxyuris vermicularis, Strongyloides stercoralis* with *Hymenolepis nana; Tri-*

churis trichiura, Ascaris lumbricoides, Oxyuris vermicularis with *Strongyloides stercoralis; Trichuris trichiura, Ascaris lumbricoides, Necator americanus* with *Hymenolepis nana.*

The youngest individual in this study found to be infected was in this institution. The child was 2 years of age, and had a quadruple infection of *Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis* and *Strongyloides stercoralis.*

The children played with dogs. There was no systematic medical inspection and the stools had never been examined before. Raw washed fruits and vegetables were eaten by the inmates of this institution. Several were dirt eaters, though they were not specified. The previous environments were unknown.

The orphan asylum designated in the tables as No. 3 presented seventy-five individuals for examination, of whom forty-two were males and thirty-three were females, their ages ranging from 4 to 17 years. Of the infected cases, twenty-eight were males and twenty females. The previous environments were not known. The parasitic infections in the forty-eight instances in this group were as follows: *Trichuris trichiura* in thirty-one, or 64.58 per cent., of the cases; *Ascaris lumbricoides* in ten, or 20.83 per cent.; *Oxyuris vermicularis* in eight, or 16.67 per cent.; *Strongyloides stercoralis* in three, or 6.25 per cent., and *Hymenolepis nana* in five, or 10.42 per cent.

The multiple infections showed: *Trichuris trichiura* with *Oxyuris vermicularis* in five instances; *Trichuris trichiura* with *Ascaris lumbricoides* in two instances; *Trichuris trichiura* with *Hymenolepis nana,* and *Ascaris lumbricoides* with *Strongyloides stercoralis* in one instance each.

There were no dogs or animals in this institution. Raw, washed fruits and vegetables were eaten here. There were no crawling children, dirt eaters or finger suckers. The stools were never examined.

The orphan asylum designated in the tables as No. 4 contributed sixty-seven stools for examination from thirty-nine males and twenty-eight females. Their ages ranged from 3 to 14 years. Examination showed twenty-four infections, twelve males and twelve females. The infections were as follows: *Trichuris trichiura* in fifteen, or 62.5 per cent.; *Ascaris lumbricoides* in five, or 20.83 per cent.; *Hymenolepis nana* in five, or 20.83 per cent., and *Necator americanus* in one, or 4.16 per cent. Double infection occurred twice with the same parasites, namely, *Trichuris trichiura* with *Hymenolepis nana.* The habits and surroundings of the inmates of this institution could not be ascertained, except that raw, washed vegetables were eaten here, and the stools had been examined once for hookworm a number of years ago.

The infant's asylum in this study is designated in the tables as

No. 5. There were 110 stools examined from children ranging in age from 3 weeks to 5 years, of whom seventy-three were males and thirty-seven were females. Of those infected, fifteen were males and six were females. No infections occurred in this institution before the second year. The youngest child was 28 months old, and was infected with *Ascaris lumbricoides*. This child subsequently died of tuberculosis. There were thirteen single infections and eight multiple infections. *Trichuris trichiura* infections were found in nine, or 42.85 per cent.; *Ascaris lumbricoides* in twelve, or 57.14 per cent.; *Oxyuris vermicularis* in three, or 14.28 per cent.; *Hymenolepis nana* in four, or 19.04 per cent., and *Hymenolepis diminuta, Strongyloides stercoralis* and *Necator americanus* in one, or 4.76 per cent, instances each. The multiple infections were: *Trichuris trichiura* with *Strongyloides stercoralis* one time; *Trichuris trichiura* with *Ascaris lumbricoides* two times, and the following combination one time each: *Ascaris lumbricoides* with *Hymenolepis nana; Trichuris trichiura* with *Hymenolepis nana; Trichuris trichiura* with *Ascaris lumbricoides* and *Hymenolepis diminuta;* and *Trichuris trichiura* with *Ascaris lumbricoides* and *Oxyuris vermicularis*. It is of interest that the rat tape worm, *Hymenolepis diminuta,* was found in this institution, and in an individual 3 years old. One of the youngest infections in this series occurred in this institution, namely, the child 28 months old infected with *Ascaris lumbricoides*.

In this institution medical inspection is frequent. The laboratory of the board of health examines stools when a child is suspected, and also makes differential blood examinations in certain conditions when called to do so. There are no dogs or cats in the institution. The children receive both washed and raw vegetables, and fruits and bananas unwashed and raw. Many of these inmates are born in the institution and those entering later come from unsanitary environment. Crawlers, finger suckers and dirt eaters are common here.

The institution designated in the tables as No. 6 is a white waif's home. The stools of fifty-one inmates were examined. All were males whose ages were not obtainable. The percentage of infections in this institution was the greatest in this study, showing forty-six infections in fifty-one individuals, or 90.2 per cent. of the inmates infected. The infections found were: *Trichuris trichiura* in twenty-nine, or 62.83 per cent. of the infections; *Ascaris lumbricoides* in fifteen, or 32.61 per cent.; *Oxyuris vermicularis* in ten, or 21.74 per cent.; *Strongyloides stercoralis* in four, or 8.69 per cent.; *Necator americanus* in four, or 8.69 per cent., and *Hymenolepis nana* in fourteen, or 13.43 per cent., of the instances. The multiple infections were as follows: *Trichuris trichiura* with *Ascaris lumbricoides* in six, or

13.1 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* and *Hymenolepis nana* in six, or 13.1 per cent.; *Trichuris trichiura* with *Hymenolepis nana* in three, or 6.52 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* and *Necator americanus* in two, or 4.36 per cent., and *Strongyloides stercoralis* with *Necator americanus, Strongyloides stercoralis* with *Hymenolepis nana,* and *Trichuris trichiura* with *Ascaris lumbricoides, Strongyloides stercoralis* and *Hymenolepis nana* in one instance each. Mutiple infections occurred in twenty, or 43.48 per cent., of the instances.

The previous environment of the inmates of this institution are rarely sanitary. The population is transient. There is no medical inspection, and while the sanitary conditions are very good, particularly in the pantry and kitchen, the origin, duration of residence and lack of medical inspection may easily be accountable for the heavy infection. There are no dogs or cats in the institution. The vegetables and fruit eaten here are washed.

In the institution designated in the tables as No. 7, the conditions are identical with those in No. 6. In No. 7 the inmates are colored waifs. The stools of seventy-two individuals were examined showing an infection second in severity only to the white waif's home, as the stools of sixty of the seventy-two inmates were infected, or an 83 per cent. infection. The parasites causing the infection were: *Trichuris trichiura* in forty-two, or 70 per cent.; *Ascaris lumbricoides* in seventeen, or 28.33 per cent.; *Hymenolepis nana* in eleven, or 18.33 per cent.; *Oxyuris vermicularis* in five, or 8.33 per cent.; *Strongyloides stercoralis* in eleven, or 18.33 per cent., and *Necator americanus* in four, or 6.66 per cent. The multiple infections were as follows: *Trichuris trichiura* with *Strongyloides stercoralis* in six, or 10 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* in six, or 10 per cent.; *Trichuris trichiura* with *Hymenolepis nana* in four, or 6.66 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* and *Strongyloides stercoralis* in two, or 3.33 per cent.; *Trichuris trichiura* with *Oxyuris vermicularis* in two, or 3.33 per cent., and one, or 1.66 per cent., instances of the following combination: *Oxyuris vermicularis* with *Strongyloides stercoralis; Oxyuris vermicularis* with *Hymenolepis nana; Ascaris lumbricoides* with *Strongyloides stercoralis* and *Hymenolepis nana; Ascaris lumbricoides* with *Necator americanus* and *Hymenolepis nana; Ascaris lumbricoides* with *Necator americanus,* and *Necatar americanus* with *Hymenolepis nana.* Multiple infections occurred in twenty-six, or 43.33 per cent., of the infections in this institution, the greatest multiple infections occurring in this study.

Age.—The ages of those whose stools were examined ranged from 3 weeks to 18 years, as follows: Under 1 year, fifty-five; 1 year

and under 2 years, seventeen; 2 years, three; 3 years, sixteen; 4 years, twenty; 5 years, twenty-one; 6 years, thirteen; 7 years, twenty-four; 8 years, thirty-five; 9 years, thirty; 10 years, thirty-nine; 11 years, forty-eight; 12 years, forty-five; 13 years, forty-two; 14 years, twenty-six; 15 years, twenty-three; 16 years, ten; 17 years, three; 18 years, two. In 123 instances the ages were not given.

There were no infections under 2 years. Two years, two, or 0.33 per cent.; 3 years, nine, or 1.51 per cent.; 4 years, ten, or 1.68 per cent.; 5 years, eight, or 1.34 per cent.; 6 years, ten, or 1.68 per cent.; 7 years, twelve, or 2.01 per cent.; 8 years, twenty-four, 4.03 per cent.; 9 years, seventeen, or 2.85 per cent.; 10 years, seventeen, or 2.85 per cent.; 11 years, twenty-seven, or 4.53 per cent.; 12 years, twenty-six, or 4.36 per cent.; 13 years, twenty-one, or 3.52 per cent.; 14 years, fifteen, or 2.52 per cent.; 15 years, eight, or 1.34 per cent.; 16 years, three, or 0.41 per cent.; 17 years, one, or 0.16 per cent.; 18 years, one, or 0.16 per cent.

Sex.—Of the 595 stools examined, 365 were from males and 230 from females. The 317 infections included 213, or 67.19 per cent. males and 104, or 32.81 per cent., females; 58.38 per cent. of the males and 45.22 per cent. of the females investigated were infected.

The number of males whose stools were examined at the various ages were: Under 1 year, thirty-five; over 1 year and under 2 years, nine; 2 years, one; 3 years, ten; 4 years, ten; 5 years, fifteen; 6 years, nine; 7 years, fourteen; 8 years, fourteen; 9 years, fourteen; 10 years, twenty-two; 11 years, twenty-three; 12 years, twenty; 13 years, eighteen; 14 years, fourteen; 15 years, eight; 16 years, six; 17 years, none; 18 years, none. Of 123 males the ages were not given.

The males infected at the given ages were: 2 years, one, or 0.47 per cent.; 3 years, six, or 2.85 per cent.; 4 years, six, or 2.85 per cent.; 5 years, six, or 2.85 per cent.; 6 years, six, or 2.85 per cent.; 7 years, eight, or 3.8 per cent.; 8 years, nine, or 4.28 per cent.; 9 years, eight, or 3.80 per cent.; 10 years, ten, or 4.74 per cent.; 11 years, fourteen, or 6.7 per cent.; 12 years, fourteen, or 6.7 per cent.; 13 years, five, or 2.37 per cent.; 14 years, seven, or 3.35 per cent.; 15 years, four, or 1.9 per cent.; 16 years, three, or 1.42 per cent. Of 106 infected males the ages were not given.

The number of females whose stools were examined at the various ages were: Under 1 year, twenty; over 1 year and under 2 years, eight; 2 years, two; 3 years, six; 4 years, ten; 5 years, six; 6 years, four; 7 years, ten; 8 years, twenty-one; 9 years, sixteen; 10 years, seventeen; 11 years, twenty-five; 12 years, twenty-five; 13 years, twenty-four; 14 years, twelve; 15 years, fifteen; 16 years, four; 17 years, three; 18 years, two.

The females infected at the various ages were: 2 years, one, or 0.31 per cent.; three years, three, or 0.95 per cent.; 4 years, four, or 1.26 per cent.; 5 years, two, or 0.63 per cent.; 6 years, four, or 1.26 per cent.; 7 years, four, or 1.26 per cent.; 8 years, fifteen, or 4.74 per cent.; 9 years, nine, or 2.84 per cent.; 10 years, seven, or 2.21 per cent.;

TABLE 1.—THE PERCENTAGES OF THE MALES AND FEMALES EXAMINED AT THE VARIOUS AGES

Age, Years	Males			Females		
	Number Examined	Number Infected	Per Cent. Infected	Number Examined	Number Infected	Per Cent. Infected
Under 1	35	0	0	20	0	0
1	9	0	0	8	0	0
2	1	1	100	2	1	50
3	10	6	60	6	3	50
4	10	6	60	10	4	40
5	15	6	40	6	2	33.3
6	9	6	66.6	4	4	100
7	14	8	57	10	4	40
8	14	9	64	21	15	71.5
9	14	8	57	16	9	56.2
10	22	10	45.4	17	7	41.2
11	23	14	61	25	13	52
12	20	14	70	25	12	48
13	18	5	27.8	24	16	66.7
14	14	7	50	12	8	66.7
15	8	4	50	15	4	26.6
16	6	3	50	4	4	100
17	0	0	0	3	2	66.7
18	0	0	0	1	1	100

In 123 instances the ages of males examined were not given.
In 106 instances the ages of males infected were not given.

TABLE 2.—SYMPTOMS MANIFESTED IN INFECTED AND NONINFECTED CASES

Symptom	Number of Cases	Per Cent. of All Cases	Number of Negative Cases	Per Cent. of Negative Cases	Number of Infected Cases	Per Cent. of Infected Cases
Abdominal pain	19	14.3	16	16.8	3	7.9
Constipation	36	27.1	33	34.8	3	7.9
Diarrhea	2	1.5	2	2.12	1	2.63
Alternating diarrhea and constipation	2	1.5	1	1.05		
Capricious appetite	2	1.5	2	2.12		
Good appetite	14	10.52	5	5.26	9	2.37
Poor appetite	7	5.25	6	6.32	1	2.63
Ravenous appetite	52	39.2	42	44.2	10	26.0
Bad breath	43	33.1	35	36.9	8	21.4
Tympanitis	0	0.0	0	0.0	0	0.0
Grinding teeth	21	15.8	14	14.7	7	18.35
Disturbed sleep	14	10.27	10	10.5	4	10.52
Nose picking	20	15.06	14	14.7	6	15.8
Pruritus	1	0.75	1	1.5	0	0.0
Bed wetting	7	5.25	3	3.18	4	10.52
Frequent urination	4	3.0	3	3.18	1	2.63
Convulsions	1	0.75	1	1.05		
Lassitude	49	36.95	38	40.0	11	28.9

11 years, thirteen, or 4.1 per cent.; 12 years, twelve, or 3.76 per cent.; 13 years, sixteen, or 5.05 per cent.; 14 years, eight, or 2.52 per cent.; 15 years, four, or 1.26 per cent.; 16 years, none; 17 years, one, or 0.31 per cent.; 18 years, one, or 0.31 per cent.

Unfortunately, more information could not be secured with regard to the symptoms. In only 133 instances was it possible to secure the symptoms. This included ninety-five negative cases and thirty-eight infected cases.

The comparative frequency of the symptoms attributable to infection with intestinal parasites in the noninfected cases with those infected in this series, even though it is small, should emphasize the necessity of finding either the ova or the parasite itself in making a diagnosis of worms before subjecting an individual, and more especially the infant and small child, to the ordeal of unnecessary medication, as the positive diagnosis is a very simple matter. Especial attention is drawn to the comparative frequency in the noninfected cases with the infected cases where nose picking, grinding of the teeth, disturbed sleep, ravenous appetite and lassitude existed.

THE PARASITES

The parasites found in this series were as follows: *Trichuris trichiura; Ascaris lumbricoides; Oxyuris vermicularis; Strongyloides stercoralis; Necator americanus; Hymenolepis nana* and *Hymenolepis diminuta.* They appeared singly and in various combinations as shown in Table 4.

Trichuris trichiura.—This parasite infected 209 individuals. It was a single offender in 120, or 57.42 per cent., cases; double in sixty-eight, or 32.54 per cent.; triple in eighteen, or 8.61 per cent., and quadruple in three, or 1.43 per cent. Its age incidence was as follows: 2 years, one, or 0.72 per cent.; 3 years, four, or 2.9 per cent.; 4 years, six, or 4.35 per cent.; 5 years, one, or 0.72 per cent.; 6 years, eight, or 5.8 per cent.; 7 years, six, or 4.35 per cent.; 8 years, eighteen, or 13.04 per cent.; 9 years, fifteen, or 10.87 per cent.; 10 years, sixteen, or 11.59 per cent.; 11 years, nineteen, or 13.77 per cent.; 12 years, sixteen, or 11.59 per cent.; 13 years, thirteen, or 9.42 per cent.; 14 years, seven or 5.07 per cent.; 16 years, four, or 2.9 per cent.; 16 years, two, or 1.45 per cent.; 17 years, one, or 0.72 per cent.; 18 years, one, or 0.72 per cent., and in seventy-one instances the ages were not given.

Its period of maximum infection was therefore from 8 to 13 years, inclusive, when over 70 per cent. of its infections occurred.

Males and females were infected at the various ages as follows: 2 years, no males and one female; 3 years, three males and one female; 4 years, five males and one female; 5 years, one male and no female; 6 years, four males and four females; 7 years, five males and one female; 8 years, six males and twelve females; 9 years, seven males and eight females; 10 years, nine males and seven females; 11 years,

eleven males and eight females; 12 years, six males and 10 females; 13 years, four males and nine females; 14 years, three males and four females; 15 years, two males and two females; 16 years, one male and one female; 17 years, no males and one female; 18 years, no males and one female.

Ascaris lumbricoides.—This parasite infected eighty-six individuals. It was a single offender in thirty-six, or 41.86 per cent., of its infections; double in twenty-eight, or 32.56 per cent.; triple in nineteen, or 22.09 per cent., and quadruple in three, or 3.49 per cent. Its age incidence was as follows: 2 years, two, or 3.7 per cent.; 3 years, four, or 7.4 per cent.; 4 years, three, or 5.6 per cent.; 5 years, five, or 9.2 per cent.; 6 years, none; 7 years, five, or 9.2 per cent.; 8 years, seven, or 13 per cent.; 9 years, four, or 7.4 per cent.; 10 years, six, or 11.1 per cent.; 11 years, five, or 9.2 per cent.; 12 years, five, or 9.2 per cent.; 13 years, five, or 9.2 per cent.; 14 years, three, or 5.6 per cent., and in thirty-two instances the ages were not given.

Its period of maximum infection was therefore from 5 to 13 years when over 77 per cent. of its infections occurred.

Males and females were infected at the various ages as follows: 2 years, one male and one female; 3 years, two males and two females; 4 years, two males and one female; 5 years, four males and one female; 7 years, five males and no females; 8 years, three males and four females; 9 years, no males and four females; 10 years, four males and two females; 11 years, three males and 2 females; 12 years, three males and two females; 13 years, two males and three females; 14 years, three males.

Oxyuris vermicularis.—This parasite infected forty-one individuals. It was a single offender in twenty-three, or 56.10 per cent., of the cases; double in fifteen, or 36.58 per cent.; triple in two, or 4.88 per cent., and quadruple in one, or 2.44 per cent. Its age incidence was as follows: 2 years, one, or 3.85 per cent.; 3 years, none; 4 years, three, or 11.54 per cent.; 5 years, one, or 3.85 per cent.; 6 years, three, or 11.54 per cent.; 7 years, two, or 7.69 per cent.; 8 years, four, or 15.38 per cent.; 9 years, one, or 3.85 per cent.; 10 years, none; 11 years, one, or 3.85 per cent.; 12 years, two, or 7.69 per cent.; 13 years, one, or 3.85 per cent.; 14 years, three, or 11.54 per cent.; 15 years, two, or 7.69 per cent.; 16 years, two, or 7.69 per cent., and in fifteen instances the ages were not given.

There was no period of maximum infection, as the parasite was found rather evenly distributed.

Males and females were infected at the various ages as follows: 2 years, no males and one female; 4 years, two males and one female; 5 years, one male and no female; 6 years, two males and one female;

7 years no males and two females; 8 years, one male, and three females; 9 years, one male and no females; 11 years, no males and one female; 12 years, two males and no females; 13 years, no males and one female; 14 years, two males and one female; 15 years, one male and one female; 16 years, two males and no females.

Strongyloides stercoralis.—This parasite infected forty individuals. It was a single offender in eight, or 20 per cent., of its infections; double in twenty-two, or 55 per cent.; triple in eight, or 20 per cent., and quadruple in two, or 5 per cent. Its age incidence was a follows: 2 years, one, or 4 per cent.; 3 years, none; 4 years, one, or 4 per cent.; 5 years, none; 6 years, one, or 4 per cent.; 7 years, three, or 12 per cent.; 8 years, one, or 4 per cent.; 9 years, three, or 12 per cent.; 10 years, four, or 16 per cent.; 11 years, two, or 8 per cent.; 12 years, three, or 12 per cent.; 13 years, three, or 12 per cent.; 14 years, one, or 4 per cent.; 15 years, two, or 8 per cent., and in fifteen instances the ages were not given.

Its period of maximum infection was therefore from 7 to 13 years, inclusive, when 76 per cent. of the infections occurred.

Males and females were infected at the various ages as follows: 2 years, no males and one female; 4 years, no males and one female; 6 years, one male and no females; 7 years, one male and two females; 8 years, one male and no females; 9 years, one male and two females; 10 years, two males and two females; 11 years, two males and no females; 12 years, three males and no females; 13 years, no males and three females; 14 years, no males and one female; 15 years, no males and two females.

Necator americanus.—This parasite infected eighteen individuals. It was a single offender in 5, or 27.78 per cent., of its infections; double in seven, or 38.89 per cent.; triple in five, or 27.78 per cent., and quadruple in one, or 5.55 per cent. Its age incidence was as follows: 2 years, none; 3 years, one, or 10 per cent.; 7 years, one, or 10 per cent.; 9 years, one, or 10 per cent.; 11 years, one, or 10 per cent.; 12 years, one, or 10 per cent., 13 years, three, or 30 per cent.; 15 years, two, or 20 per cent., and in eight instances the ages were not given.

No period could be considered as of maximum infection with this parasite.

Males and females were infected at the various ages as follows: 3 years, no males and one female; 7 years, one male and no females; 9 years, one male and no females; 11 years, no males and one female; 12 years, no males and one female; 13 years, one male and two females; 15 years, no males and two females.

Hymenolepis nana.—This parasite infected fifty-five individuals

TABLE 3.—Results of One Examination of Feces for Animal Parasites

Institution	Number Examined	Number of Infections		Single Infection		Double Infection		Triple Infection		Quadruple Infection		Trichuris Trichiura		Ascaris Lumbricoides		Oxyuris Vermicularis		Strongyloides Stercoralis		Necator Americanus		Hymenolep Nana		Hymenolepis Diminuta	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
No. 1	95	17	17.89	15	15.78	2	2.1	0	0.0	0	0.0	10	10.0	1	1.05	6	6.31	0	0.0	0	0.0	2	2.1	0	0.0
No. 2	125	101	80.08	62	49.60	30	24.0	7	5.68	2	1.6	73	58.40	26	20.80	9	7.20	21	16.80	8	6.40	14	11.20	0	0.0
No. 3	75	48	64.0	39	52.0	9	12.0	0	0.0	0	0.0	31	41.33	10	13.33	8	10.66	3	4.0	0	0.0	5	6.66	0	0.0
No. 4	67	24	35.82	22	32.83	2	2.98	0	0.0	0	0.0	15	22.37	5	7.46	0	0.0	0	0.0	1	1.49	5	7.46	0	0.0
No. 5	110	21	19.09	13	11.81	6	5.45	2	1.81	0	0.0	9	8.18	12	10.9	3	2.72	1	0.9	1	0.9	4	3.63	1	0.9
No. 6	51	46	90.2	26	50.98	11	21.58	8	15.88	1	1.96	29	56.86	15	29.41	10	19.60	4	7.84	4	7.84	14	27.45	0	0.0
No. 7	72	60	83.33	34	47.22	22	30.55	4	5.55	0	0.0	42	58.34	17	23.61	5	6.94	11	15.27	4	5.55	11	15.27	0	0.0
Total	595	317	53.27	211	35.47	82	13.78	21	3.53	3	0.5	209	35.12	86	14.62	41	6.89	40	6.72	18	3.0	55	9.25	1	0.17

TABLE 4.—Results of Second Examination of Feces for Animal Parasites

Institution	Number Infected	Single Infection		Double Infection		Triple Infection		Quadruple Infection		Trichuris Trichiura		Ascaris Lumbricoides		Oxyuris Vermicularis		Strongyloides Stercoralis		Necator Americanus		Hymenolepis Nana		Hymenolepis Diminuta	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
No. 1	17	15	88.25	2	11.75	0	0.0	0	0.0	10	58.82	1	5.88	6	35.29	0	0.0	0	0.0	2	11.76	0	0.0
No. 2	101	62	61.39	30	29.7	7	6.93	2	1.98	73	72.29	26	25.34	9	8.91	21	20.89	8	7.92	14	13.86	0	0.0
No. 3	48	39	81.25	9	18.75	0	0.0	0	0.0	31	64.58	10	20.83	8	16.67	3	6.25	0	0.0	5	10.42	0	0.0
No. 4	24	22	91.66	2	8.33	0	0.0	0	0.0	15	62.5	5	20.83	0	0.0	0	0.0	1	4.16	5	20.83	0	0.0
No. 5	21	13	61.90	6	28.57	2	9.52	0	0.0	9	42.85	12	57.14	3	14.28	1	4.76	1	4.76	4	19.04	1	4.76
No. 6	46	26	56.52	11	23.91	8	17.39	1	2.17	29	62.83	15	32.61	10	21.74	4	8.69	4	8.69	14	13.43	0	0.0
No. 7	60	34	56.66	22	36.66	4	6.66	0	0.0	42	70.0	17	28.33	5	8.33	11	18.33	4	6.66	11	18.33	0	0.0
Total	217	211	66.56	82	25.87	21	6.62	3	0.95	209	65.9	86	27.13	41	12.93	40	12.62	18	5.68	55	17.35	1	0.32

It was a single offender in nineteen, or 34.55 per cent., of its infections; double in twenty-four, or 43.64 per cent.; triple in ten, or 18.18 per cent., and quadruple in two, or 3.63 per cent. Its age incidence was as follows: 3 years, three, or 10 per cent.; 4 years, one, or 3.33 per cent.; 5 years, three, or 10 per cent.; 6 years, one, or 3.33 per cent.; 7 years, one, or 3.33 per cent.; 8 years, two, or 6.67 per cent.; 9 years, one, or 3.33 per cent.; 11 years, five, or 16.67 per cent.; 12 years, five, or 16.67 per cent.; 13 years, two, or 6.67 per cent.; 14 years, five, or 16.67 per cent., and 16 years, one, or 3.33 per cent. In twenty-five instances the ages were not given.

In the infection of this parasite there also seemed no period of maximum infection, though 70 per cent. of the infections occurred above the age of 8 years.

Males and females were infected at the various ages as follows: 3 years, three males and no females; 4 years, one female; 5 years, two males and one female; 6 years, one male and no females; 7 years, one male and no females; 8 years, one male and one female; 9 years, no males and one female; 11 years, three males and two females; 12 years, four males and one female; 13 years, no males and two females; 14 years, one male and four females; 16 years, one male and no females.

Hymenolepis diminuta.—This parasite was found in one instance in which there was a triple infection with *Trichuris trichiura* and *Ascaris lumbricoides*. The individual infected was a female, 3 years of age.

BLOODS

The bloods of 307 individuals included in this study were examined. Differential counts were made from the bloods of 124 of the negative cases and 183 of the positive cases. Table 5 gives the numbers and averages of bloods for the different ages for both positive and negative cases, and the average of all the bloods of the negative cases as well as the average of all the bloods of the positive cases. A comparison of the average differential count for all ages shows an increase in the eosinophils of the infected cases of 3.5 per cent. over the average of the noninfected cases, the increase apparently being at the expense of the neutrophils.

In the study of the eosinophils for the averages at the different ages, the variations in negative cases were from 0 to 4.8 per cent. In only three instances, namely, for the ages of 4, 8 and 11 years, respectively, were the averages above 4 per cent., and in each of these instances the average was heightened by one or two cases with marked eosinophilia. On the other hand, the averages for the different ages in the positive cases, with the exception of the eighteenth year, where only one case was examined, and where tthe eosinophils were 2 per

TABLE 5.—NUMBER OF INFECTED AND NONINFECTED MALES AND FEMALES OF VARIOUS AGES

Age	Institution 1		2		3		4		5		Total	
	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected
Under 1 year												
Male	..	..	..	..	..	..	..	..	..	35	..	35
Female	..	..	..	..	..	..	..	..	..	20	..	20
1 year												
Male	..	..	..	..	..	..	..	..	..	9	..	9
Female	..	1	..	..	..	..	..	..	..	7	..	8
2 years												
Male	..	..	..	..	..	..	..	..	1	..	1	..
Female	..	..	1	..	..	..	..	..	..	1	1	1
3 years												
Male	..	..	..	..	..	..	1	..	5	4	6	4
Female	..	1	..	1	..	..	..	..	3	1	3	3
4 years												
Male	..	..	1	..	..	2	..	..	5	2	6	4
Female	..	1	1	1	1	1	..	1	2	2	4	6
5 years												
Male	..	1	..	..	2	..	..	1	4	7	6	9
Female	1	..	..	2	..	..	..	1	1	1	2	4
6 years												
Male	1	2	3	..	2	..	..	1	..	..	6	3
Female	..	..	1	..	3	..	..	..	..	..	4	..
7 years												
Male	..	3	5	..	1	..	2	3	..	..	8	6
Female	..	1	2	..	1	1	1	4	..	..	4	6
8 years												
Male	..	1	3	..	5	1	1	3	..	..	9	5
Female	1	5	9	1	4	2	1	0	..	..	15	6
9 years												
Male	..	1	7	..	..	1	1	4	..	..	8	6
Female	..	4	6	1	1	1	2	1	..	..	9	7
10 years												
Male	1	5	4	..	4	2	1	5	..	..	10	12
Female	..	4	6	2	..	2	1	2	..	..	7	10
11 years												
Male	2	4	3	..	6	1	3	4	..	..	14	9
Female	2	3	8	3	3	2	..	4	..	..	13	12
12 years												
Male	2	3	6	..	4	3	2	1	..	..	14	6
Female	1	5	6	4	2	2	3	2	..	..	12	13
13 years												
Male	..	5	3	..	1	3	1	5	..	..	5	13
Female	1	5	9	3	3	..	3	..	..	..	16	8
14 years												
Male	2	5	3	..	2	2	..	..	..	..	7	7
Female	..	3	7	..	..	..	1	1	..	..	8	4
15 years												
Male	..	4	3	..	1	..	..	..	..	..	4	4
Female	1	7	2	3	1	1	..	..	..	..	4	11
16 years												
Male	1	3	2	..	..	..	..	..	..	..	3	3
Female	..	2	..	1	1	1	..	..	..	..	..	4
17 years												
Male	..	..	..	..	..	..	..	..	..	..	..	..
Female	..	..	..	2	1	..	..	..	..	..	1	2
18 years												
Male	..	..	..	..	..	..	..	..	..	..	..	..
Female	1	1	..	..	..	..	..	..	..	..	1	1
Totals	17	78	101	24	48	27	24	43	21	89	211	261

cent., ranged from 4.7 to 9.2 per cent., clearly showing the value of the eosinophilia as an aid in the diagnosis of intestinal parasitic infections.

Further interest is evidenced in the study of the eosinophils of the individual cases. Of the infected cases in three instances there was a 28 per cent. eosinophilia; in one instance there was a 23 per cent. eosinophilia; in one a 22 per cent., in two an 18 per cent., in one a 17 per cent., in one a 16 per cent., in one a 15 per cent., in eight a 14 per cent., in three a 13 per cent., in seven a 12 per cent., in five an 11 per cent., in seven a 10 per cent., in eleven a 9 per cent., in eight an 8 per cent., in twelve a 7 per cent., in fifteen a 6 per cent., in nine a 5 per cent. and in fourteen a 4 per cent. eosinophilia; or in 59.5 per cent. the percentage of eosinophils was four or more; in 51.9 per cent. it was five or more; in 47 per cent., six or more; in 38.7 per cent., seven or more; in 32.2 per cent., eight or more; in 27.8 per cent., nine or more; in 21.8 per cent., 10 or more; in 18 per cent., 11 or more; in 15.3 per cent., 12 or more; in 11.4 per cent., 13 or more; in 9.8 per cent., 14 or more; in 5.4 per cent., 15 or more; in 4.9 per cent., 16 or more; in 4.3 per cent., 17 or more; in 3.8 per cent., 18 or more; in 2.7 per cent., 22 or more; in 2.1 per cent., 23 or more, and in 1.6 per cent. of the infected cases there was a 28 per cent. eosinophil count.

In the noninfected cases in one instance there was an 18 per cent. eosinophilia; in one a 16 per cent. eosinophilia; in two a 13 per cent.; in one a 12 per cent.; in one a 10 per cent.; in four an 8 per cent.; in four a 7 per cent.; in four a 6 per cent.; in five a 5 per cent., and in seventeen a 4 per cent. eosinophilic count; or, in 32 per cent. of the cases the eosinophils numbered 4 per cent. or more; in 18.5 per cent., 5 or more; in 14.5 per cent., 6 or more; in 11.2 per cent., 7 or more; in 8 per cent., 8 or more; in 4.8 per cent., 10 or more; in 4 per cent., 12 or more; in 3.2 per cent., 13 or more; in 1.6 per cent., 16 or more, and in 0.8 per cent. of the noninfected cases there was an 18 per cent. eosinophil count. These results are of especial interest, because, while an eosinophilia should suggest an intestinal parasitic infection, it is not pathognomonic of it.

It is of interest to note that of the positive cases all the eosinophilias from 15 per cent. and above were in the same institution (No. 2).

In studying the eosinophils in their relattion to the infections of the various parasites singly and in combination, the following was revealed: The maximum eosinophilia in the single infection by the *Trichuris trichiura* was 15 per cent., which occurred in one instance. While, on the other hand, there were two infections by this parasite in which there were no eosinophils shown in the count. The eosinophil count was 4 per cent. or less, as follows: In fourteen instances

the percentage was 1 per cent.; in nine it was 2 per cent.; in six it was 3 per cent., and in seven it was 4 per cent.

The maximum eosinophilia in connection with the single infection with the *Ascaris lumbricoides* was 16 per cent.; the minimum 0.0 per cent. The eosinophil count was 4 per cent. or less, as follows: One patient had a 1 per cent., four patients had a 4 per cent., two patients had a 3 per cent., and three patients had a 4 per cent.

TABLE 6.—PARASITES AND NUMBER OF TIMES EACH WAS FOUND ALONE OR ASSOCIATED WITH OTHERS

Infections	Institutions							Total
	1	2	3	4	5	6	7	
Trichuris trichiura	8	40	23	13	3	11	22	120
Ascaris lumbricoides	0	12	7	5	6	0	6	36
Oxyuris vermicularis	6	2	3	0	1	10	1	23
Strongyloides stercoralis	0	6	2	0	0	1	1	8
Necator americanus	0	1	0	1	1	1	1	5
Hymenolepis nana	1	3	4	3	2	3	3	19
Hymenolepis diminuta	0	0	0	0	0	0	0	0
Trichuris trichiura, Ascaris lumbricoides	1	6	2	0	2	6	6	23
Trichuris trichiura, Oxyuris vermicularis	0	3	5	0	0	0	2	10
Trichuris trichiura, Strongyloides stercoralis	0	8	0	0	1	0	6	15
Trichuris trichiura, Necator americanus	0	3	0	0	0	0	0	3
Trichuris trichiura, Hymenolepis nana	1	5	1	2	1	3	4	17
Ascaris lumbricoides, Oxyuris vermicularis	0	0	0	0	1	0	0	1
Ascaris lumbricoides, Strongyloides stercoralis	0	0	1	0	0	0	0	1
Ascaris lumbricoides, Necator americanus	0	0	0	0	0	0	1	1
Ascaris lumbricoides, Hymenolepis nana	0	1	0	0	1	0	0	2
Oxyuris vermicularis, Strongyloides stercoralis	0	1	0	0	0	0	1	2
Oxyuris vermicularis, Hymenolepis nana	0	1	0	0	0	0	1	2
Strongyloides stercoralis, Necator americanus	0	1	0	0	0	1	0	2
Strongyloides stercoralis, Hymenolepis nana	0	1	0	0	0	1	0	2
Necator americanus, Hymenolepis nana	0	0	0	0	0	0	1	1
Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis	0	0	0	0	1	0	0	1
Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis	0	4	0	0	0	0	2	6
Trichuris trichiura, Ascaris lumbricoides, Necator americanus	0	1	0	0	0	2	0	3
Trichuris trichiura, Ascaris lumbricoides, Hymenolepis nana	0	0	0	0	0	6	0	6
Trichuria trichiura, Ascaris lumbricoides, Hymenolepis diminuta	0	0	0	0	1	0	0	1
Trichuris trichiura, Necator americanus, Hymenolepis nana	0	1	0	0	0	0	0	1
Ascaris lumbricoides, Strongyloides stercoralis, Hymenolepis nana	0	0	0	0	0	0	1	1
Ascaris lumbricoides, Necator americanus, Hymenolepis nana	0	0	0	0	0	0	1	1
Oxyuris vermicularis, Strongyloides stercoralis, Hymenolepis nana	0	1	0	0	0	0	0	1
Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis, Strongyloides stercoralis	0	1	0	0	0	0	0	1
Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, Hymenolepis nana	0	0	0	0	0	1	0	1
Trichuris trichiura, Ascaris lumbricoides, Necator americanus, Hymenolepis nana	0	1	0	0	0	0	0	1

The *Oxyuris vermicularis* showed a maximum of 11 per cent. eosinophils in single infections and a minimum of 0.0 per cent. The eosinophil count was 4 per cent. or less, as follows: In four instances there was one case with 1 per cent.; three cases with 2 per cent.; two cases with 3 per cent., and two cases with 4 per cent.

The *Strongyloides stercoralis* in single infection showed a 14 per cent. maximum eosinophil count and a 0.0 per cent. minimum count.

The eosinophil count was 4 per cent. or less, as follows: There was one case with 1 per cent., and one case with 2 per cent.

The *Necator americanus* occurred only in two instances in single infection, once with a 12 per cent. and the other time with a 13 per cent. eosinophil count. It is of interest to note that the lowest eosinophil counts with this parasite in combination under 4 per cent. were 1 per cent. with a mixed infection with *Trichuris trichiuria* and *Hymenolepis nana* in one instance, and in a double infection with *Strongyloides stercoralis* in which the eosinophil count was 3 per cent.

Hymenolepis nana showed in a single infection a maximum of 18 per cent. eosinophils, and in three instances the percentage was zero. The eosinophil count was 4 per cent. or less, as follows: There were three instances of 1 per cent., and one of 2 per cent.

TABLE 7.—Results of Examination of Blood in Infected and Noninfected Cases

Age	Not Infected						Infected					
	No. Cases	Neutrophils, per Cent.	Small Mononuclears, per Cent.	Large mononuclears, per Cent.	Eosinophils, per Cent.	Basophils, per Cent.	No. Cases	Neutrophils, per Cent.	Small Mononuclears, per Cent.	Large mononuclears, per Cent.	Eosinophils, per Cent.	Basophils, per Cent.
1 yr.	1	44.0	56.0	—	—	—	0	—	—	—	—	—
2 yrs.	0	....	....	—	—	—	1	40.0	45.0	5.0	9.0	1
3 yrs.	2	56.5	42.5	0.5	0.5	2	7	47.6	39.9	6.0	6.3	2
4 yrs.	5	55.2	38.8	0.1	5.0	0	10	50.5	39.3	5.5	4.7	0
5 yrs.	3	52.7	42.3	0.2	3.0	0	8	53.4	36.4	4.1	5.7	4
6 yrs.	2	59.0	35.0	2.5	3.5	0	10	58.2	32.1	2.1	7.0	6
7 yrs.	5	62.8	32.4	1.8	2.8	2	9	57.7	31.0	1.3	9.3	2
8 yrs.	8	57.1	35.6	2.3	4.8	2	22	57.3	35.4	9.0	6.3	1
9 yrs.	8	57.9	35.6	3.3	3.1	1	13	57.0	33.2	1.2	8.4	2
10 yrs.	13	57.6	37.6	1.8	3.0	0	15	63.0	30.3	1.1	5.5	1
11 yrs.	13	60.6	32.6	2.5	4.2	1	23	57.8	35.3	2.1	4.7	1
12 yrs.	15	63.4	32.1	2.1	2.3	1	21	57.2	23.7	2.2	6.5	4
13 yrs.	16	61.1	36.0	1.4	1.4	1	16	54.9	36.4	1.0	7.6	1
14 yrs.	10	58.4	37.3	1.5	2.8	0	14	58.3	35.8	1.0	4.8	1
15 yrs.	15	63.1	33.5	1.4	1.9	1	7	55.6	38.4	6.0	5.3	1
16 yrs.	7	60.8	35.4	2.4	1.4	0	3	57.4	32.6	3.0	7.0	0
17 yrs.	0	....	....	....	....	..	3	62.0	28.0	2.0	8.0	0
18 yrs.	1	66.0	32.0	0.2	0.0	0	1	72.0	25.0	1.0	2.0	0
Aver. for all cases	124	59.92	35.39	1.9	2.71	0.08	183	56.27	35.26	2.01	6.27	0.19

In only one case was the *Hymenolepis diminuta* found, in which instance it was in a triple infection with *Trichuris trichiura* and *Ascaris lumbricoides*. The eosinophil count in this case was 13 per cent.

The maximum eosinophil count in the double infection was 28 per cent. in two instances, both being combined infections with the *Trichuris trichiura* and *Strongyloides stercoralis*. The minimum eosinophilia in this combination was 1 per cent. in one instance. There were no others below 6 per cent.

The minimum eosinophil count in a double infection was zero in a mixed infection of *Trichuris trichiura* with *Ascaris lumbricoides*.

In two double infections the count was 1 per cent., namely, *Trichuris trichiura* with *Ascaris lumbricoides*, and *Trichuris trichiura* with *Oxyuris vermicularis*. In four instances there was a 2 per cent.; in two instances a 3 per cent., and in three instances a 4 per cent. eosinophil count.

In the triple infections the maximum eosinophil count was 28 per cent. in a combined infection of *Trichuris trichiura, Ascaris lumbricoides* and *Strongyloides stercoralis*. The minimum count was zero with an infection of *Trichuris trichiura, Ascaris lumbricoides* and *Oxyuris vermicularis;* and 1 per cent. in a triple infection of *Trichuris trichiura, Necator americanus* and *hymenolepis nana*.

The blood was examined in two quadruple infections, the eosinophil counts being 9 and 8 per cent., respectively. In the former the combined infections were of the *Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis* and *Strongyloides stercoralis*, and in the latter *Trichuris trichiura, Ascaris lumbricoides, Necator americanus* and *Hymenolepis nana*. It is of especial interest that in the former infection the individual was a child, 2 years of age.

CONCLUSIONS

1. In this study the frequency of the infections by the various parasites were: *Trichuris trichiura, Ascaris lumbricoides, Hymenolepis nana, Oxyuris vermicularis, Strongyloides stercoralis, Necator americanus* and *Hymenolepis diminuta*.

2. The hygiene, general care, duration of residence in the institution, previous environment, association with dogs, eating of dirt, absence of systematic medical inspection, seemed to have a direct bearing on the frequency of intestinal parasitic infection.

3. The two institutions where systematic medical inspections were made, even though there were many crawlers in one of these institutions, showed the lowest percentages of infections, namely, 17.89 per cent. and 19.00 per cent., respectively.

4. The two institutions with the heaviest infections were those where the population was transient and where there were no medical inspections, the infections being 90.2 and 83.33 per cent., respectively.

5. The youngest individual infected was in the least sanitary of the institutions investigated, this child was 2 years of age and had a quadruple infection of *Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis,* and *Strongyloides stercoralis*.

6. The relation of age to infection seemed negligible except that there were no infections among the infants; in other words, before the period of exposure there were no infections. The maximum number of infections occurred at the age of 11 years.

7. Males were infected over 13 per cent. more frequently than were females. Again the element of exposure seems to have had some influence. The maximum ages of the infections in boys were 11 and 12 years, and for girls, 13 years. Infections, however, were rather generally distributed over several years.

8. No information of value was obtained in studying the incidence at the different ages, except that the greatest incidence in boys was fourteen infections in twenty boys, or 70 per cent. of infections at the age of 12 years, and for the girls fifteen in twenty-one, or 71.5 per cent. of infections at the age of 8 years.

9. The symptoms usually attributed to "having worms" were as frequent, and some a little more frequent, in those not infected than in those infected.

10. One infection with the rat tape worm (*Hymenolepis diminuta*) was found in a female child, 3 years of age. The infection occurred in a triple infection of *Hymenolepis diminuta* and *Trichuris trichiura* with *Ascaris lumbricoides*.

11. An eosinophilia is a valuable aid in the diagnosis of infection with intestinal parasites. It is, however, not pathognomonic of, and its absence does not exclude, an infection.

12. The eosinophils were found as high as 28 per cent. in the positive cases, and 18 per cent. in the negative cases. They were 4 per cent. or above in 59.5 per cent. of the positive cases, and 4 per cent. above in 32 per cent. of the negative cases. It was of interest that all the eosinophilias of 15 per cent. and above in the positive cases occurred in the same institution.

13. No parasite in this study seemed to have a definite eosinophilia.

14. It was shown that the infection by any of the intestinal parasites in this study could exist with an absence of eosinophils. This was found in their single infections with the exception of *Necator americanus* and *Hymenolepis diminuta*. *Necator americanus* was found in single infections in only two cases, in which instances the eosinophils were 12 and 13 per cent., respectively. However, in mixed infection of this parasite with *Trichuris trichiura* and *Hymenolepis nana* the eosinophils were as low as 1 per cent.

The other exception was the *Hymenolepis diminuta* which was not found in single infection, but in combination with *Trichuris trichiura* and *Ascaris lumbricoides* in which case the eosinophils numbered 13 per cent.

15. Minimum eosinophil counts in double and triple infections were zero, while the maximum was 28 per cent. in each.

16. The two quadruple infections in which blood examinations were done showed an 8 and 9 per cent. eosinophilia, respectively.

17. The diagnosis of intestinal parasitic infection is an extremely easy matter. The symptoms usually ascribed to worms are as frequent in those not infected as in those infected. Blood examinations showing eosinophilias are valuable diagnostic aids, but the only way to make a positive diagnosis is by either finding the parasite or the ova in the stool of the individual. Vermifuges should, therefore, not be given to the defenseless child unless it is known that an infection exists and its character is ascertained when the specific drug may be given to combat the infection.

PROGRESS IN PEDIATRICS

REVIEW OF RECENT LITERATURE ON THE NEW-BORN

N. O. PEARCE, M.D.
Instructor in Pediatrics, University of Minnesota
CHILDREN'S CLINIC OF MINNEAPOLIS

PYLORIC STENOSIS IN INFANCY

So large an amount of discussion has appeared in the recent medical literature concerning the condition of hypertrophic pyloric stenosis, that we have selected only such articles for discussion as seem to add something to our knowledge of the condition or its treatment. Nearly all we know of the disease in infancy, has been contributed since 1897. Reuben[192] states that up to 1904, only 123 cases had appeared in the literature, while up to the time he wrote, 1914, more than one thousand cases had been reported. In his analysis there were four males affected to one female. The child is very often the first born, and its birth weight is apt to be rather above than below normal. Holt[193] calls attention to the fact that the affection nearly always occurs in breast-fed infants.

Most of our authorities agree that the pathologic change probably sets in during fetal life, developing slowly until birth, at which time it is accentuated by the stimulation of food in the stomach. Reuben does not believe that the hyperchlorhydria, which is often present, plays any part in the etiology. An attempt was made by Wachenheim,[194] to show that the pyloric hypertrophy is normally found in the new-born infant. While his series of six necropsy reports is too small for his positive findings to be of much value, he directs attention to the fact that in four consecutive necropsies of later date, on babies dying under the age of one month from causes not associated with pyloric obstruction, there was found hypertrophy of the pyloric wall.

Little progress has been made, however, in the study of the etiology of the disease. Downes[195] alleges that edema plays a very important

192. Reuben, Mark S.: Pyloric Stenosis in Infancy, Arch. Pediat. **31**:809, 1914.

193. Holt, L. Emmett: Medical Aspects of the Treatment of Pyloric Stenosis in Infancy, Am. J. Obst. **69**:1095, 1914.

194. Wachenheim, F. L.: Pyloric Hypertrophy in New-Born Infants, Am. J. Dis. Child. **10**:87 (July) 1915.

195. Downes, William A.: Operative Treatment of Pyloric Obstruction in Infants, Surg., Gynec. & Obst. **22**:251 (March) 1916.

rôle and is the factor that determines the onset of symptoms, there being present at birth a true malformation consisting of an abnormal thickening of the circular muscle fibers of the pylorus. The effort then necessary to force food through the narrowed and elongated pyloric lumen produces a circulatory disturbance, resulting in edema. The spasm is then the result and not the cause of the hypertrophy. Going a step further, Scudder,[196] in 1915, maintained that spasm had nothing whatever to do with a stenosis of the pylorus, and that the omission of its hypothetical existence from any consideration in these cases would result in an earlier adequate surgical relief. His view appears to be unsupported by other writers, however, Sill maintains that the abrupt onset of symptoms indicates that pylorospasm plays an important part in all types of cases. Those children who suffer a great deal of pain are classed as cases of spasm without stenosis by Kelley,[197] while those who throw up after eating without any pain whatever are classed as hypertrophic cases. In 1914 Holt[193] took the stand that definite persistent spasm of the pylorus without hypertrophy was yet to be proven, and no sharp division into spasmodic and hypertrophic types was possible. Another very interesting theory has been developed by Haas;[198] that there is a syndrome made up of a hypertonicity of the skeletal muscles and a tendency to spasm of the unstriated muscular system, when accompanied by psychic or physical irritation. Included in this class of patients is a large percentage of cases of pylorospasm. The congenital origin of the disease is not admitted by all investigators, however. Rachford[199] reasons that the thickening of the circular muscle fibers of the pylorus must be due to some pathologic factor, which causes the normal muscles to hypertrophy, since it has been shown that they soon return to normal size, following the Rammstedt operation. He suggests "infantile pyloric stenosis" as a more appropriate term than "congenital pyloric stenosis." Richter[200] has found no accompanying congenital malformations, although in several cases other members of the same family have died from what was apparently the same condition.

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196. Scudder, Charles L.: The Significance of Roentgen-Ray Examination Following Operation for Congenital Pyloric Tumor, Boston M. & S. J. **172**: 166, 1915.

197. Kelley, S. W.: Surgical Treatment of Pyloric Obstruction in Children, Am. J. Obst. **71**:852, 1915.

198. Haas, Sidney V.: Congenital Hypertrophic Pyloric Stenosis; a Manifestation of the Hypertonic Infant, N. Y. Acad. Med., Dec. 12, 1918.

199. Rachford, B. K.: Pyloric Stenosis in Infancy, Arch. Ped. **34**:803 (Nov.) 1917.

200. Richter, H. M.: Congenital Pyloric Stenosis; Study of Twenty-Two Cases with Operation, J. A. M. A. **62**:353 (Jan. 31) 1914.

The lesion at the pylorus is variously described by Haggard,[201] Strauss,[202] Downes,[195] Richter[200] and others. At necropsy there is found a napkin-ringlike tumor, often as hard as cartilage, that projects into the duodenum like the cervix of the uterus. It is covered by smooth, glistening peritoneum; it is always sharply defined and freely movable, and appears to be a true muscular hypertrophy. When the pyloric region is exposed to view in life, one may readily see the intermittent peristaltic contractions of the tumor independent of any of the contractions of the stomach. As it relaxes it becomes pink and soft; as it contracts its becomes white and cartilagelike. In babies three or four weeks old, the tumor is much smaller than in older patients, the size of the tumor being in direct relation to the size of the child. At the age of from seven to twelve weeks the tumor may be three or four times as large as at one month; however, its magnitude is not indicative of the amount of stenosis.

Ladd[203] differentiates between the hard, inelastic tumor of severe types of hypertrophic stenosis, which scarcely bleeds at all when incised, and the soft elastic tumor of pylorospasm, which, although at times as large as that of a true stenosis, bleeds freely when cut, and in which the mucosa does not separate from the muscular layers freely as it has been shown to do in the more severe type. Haggard[201] observes that the stomach may become ballooned as the condition advances or it may be smaller than normal, the entire walls becoming hypertrophied.

The operation of gastro-enterostomy used in the early treatment of the disease apparently did not lead to the disappearance of the tumor, as evidenced by cases reported by Haggard,[201] Walton[204] and Scudder[196] in which it remained as marked, and the stenosis as complete as ever from seven to nine months after operation. With the Rammstedt procedure, or modifications of it, the result is a normal, healthy, muscular tissue. A patient dying from enlarged thymus was seen at necropsy by Wolley[205] seven months after a Rammstedt operation had been done. At the site of the incision a linear scar 2 cm. long was found. There was no distortion or constriction and the pylorus evidently was functioning quite normally. He calls attention, as does

201. Haggard, William D.: Hypertrophic Pyloric Stenosis in Infancy, J. A. M. A. **71**:810 (Sept. 7) 1918.

202. Strauss, A. A.: Congenital Pyloric Stenosis, J. A. M. A. **71**:807 (Sept. 7) 1918.

203. Ladd, W. E.: Congenital Pyloric Stenosis, Boston M. & S. J. **178**: 767 (June 6) 1918.

204. Walton, A. J.: Congenital Pyloric Stenosis, Am. J. Surg. **15**:342, 1914.

205. Woolley, Paul G.: Operative Cure, Congenital Pyloric Stenosis, J. A. M. A. **68**:1543 (May 26) 1917.

Rachford,[199] to the frequent coincidence of enlarged thymus and pyloric stenosis.

The classical symptoms agreed on by most authorities are: (1) Persistent, recurrent, explosive vomiting; (2) emaciation; (3) visible gastric peristalsis; (4) starvation stools; (5) diminished quantity of urine; (6) bulging epigastrium with sunken lower abdomen; (7) progressive loss of weight; (8) no increase in temperature; (9) palpable tumor. The child is usuallly good, suffering from hunger only at the onset of the disease, a loss of appetite following. The onset is abrupt and occurs within the first few weeks after birth, Reuben[192] classified the time of its appearance as follows: 1 to 4 days after birth, 25 per cent.; 4 to 14, 25 per cent.; 2 to 3 weeks after birth, 25 per cent.; 3 to 6 weeks after birth, 25 per cent. The peristaltic waves are most clearly visible directly after nursing. The palpation of the tumor seems to be largely a matter of personal equation, and not essential to the diagnosis of the disease. It is best felt when the child is asleep or nursing. Kerley[206] and Richter[200] state that the tumor has been palpable in all but one case in each of their series reported, while Reuben[192] has been successful in locating it in only 25 per cent. of his cases. According to Ladd[203] the deep and high situation of the pylorus under the edge of the liver makes it impossible in many cases for anyone to feel the tumor.

Emphasis has been laid on the value of the fluoroscope and a series of roentgenograms in the diagnosis of the condition. Bismuth pills, coated with keratin, and prepared in different sizes are recommended by Hess[207] to be used in the examination. By this means Strauss[202] classified each case immediately as medical or surgical. The baby must not be placed on its back or the bismuth will accumulate on the left side and no stimulation of the peristaltic waves occur. If the child is rotated toward the right side, almost onto the abdomen, one may notice the waves in the pyloric antrum and the pylorus. A small amount of bismuth is squirted through, and then the pylorus clamps down tightly. Immediately peculiar and characteristic snakelike rythmic peristaltic contractions can be seen in the pylorus which are independent of the rest of the stomach. At the end of two hours the examination is repeated to see if anything has passed through and again at the end of four hours. Roentgenograms are also made at the last two examinations. If one half or more of the stomach contents remains at the end of four hours he considers the case surgical, if

206. Kerley, C. G.: Twenty-Six Cases of Pyloric Stenosis in Private Practice with Operation by Rammstedt Method, Arch. Pediat., June, 1918.

207. Hess, A. F.: Use of Bismuth Pills in Fluoroscopic Examination of Infant's Stomach, Am. J. Dis. Child. **9**:461 (June) 1915.

70 per cent. of the contents have passed through the pylorus, the patient can, as a rule, be cured medically. Sedgwick[208] also calls attention to the fact that in many cases with typical symptoms a patent pylorus may be shown by roentgen ray. The examination should be used in all doubtful cases.

A method of examining the pylorus by catheter is explained by Howell.[209] He makes use of the ordinary urethral catheter, 42 cm. long, in sizes from 13 F. to 22 F. and not too soft. It is to be introduced slowly and steadily in order to observe the reflexes as they occur, as they are the most important guides to a successful passage. The three reflexes are: a gagging of the infant when the tube passes into the pharynx, into the cardia, and again as it enters the pylorus. The manipulator may tell when the tube has passed the pylorus successfully by watching these reflexes as they occur, and the relief from gagging after its passage, also by the feel of the catheter, as there will be increased resistance and a tendency on the part of the tube to buckle when it passes into the narrower canal. Also if the tube is clear, when it enters the duodenum there will be a rush of gas, and sometimes bile. The appearance of bile is definite. Howell has used the duodenal catheter in cases where vomiting, visible peristalsis, no palpable or a questionable tumor, stationary weight and small digested stool appear, to determine whether or not there was stenosis at the pylorus. In cases of pylorospasm the catheter will pass with increased reflexes, and the patient may be treated by stomach washings and proper diet, but in cases of hypertrophy it will fail to pass or a small one will pass with difficulty, and the patient should be treated surgically. In commenting on this method Pieser[210] states that the only way of knowing that the catheter has passed into the duodenum is by the use of the roentgen ray.

It is generally agreed that in expert hands the prognosis in this condition is good, while treatment by the unskilled, whether medical or surgical, is still resulting in a high mortality. Mild cases may improve within a few days, but a severe case may be very protracted. Sudden deaths have been known to occur in fairly will nourished patients, where the vomiting had practically ceased and where there was no assignable cause. Of sixty-eight fatal cases collected by Ibraham,[192] eight patients died at one month, twenty-seven at two months, twenty-seven at three months, three at four months, and

208. Sedgwick, J. P.: Pylorospasm in Infants, Jour. Lancet, 1916.

209. Howell, W. W.: Diagnosis of Pyloric Stenosis and Pyloric Spasm, Arch. Pediat. **34**:199 (March) 1917.

210. Pieser, J.: Treatment of Pylorospasm in Infants, Monatschr. f. Kinderh. **13**:3, 1914.

three at five months of age. As yet, our best authorities do not all agree as to the best method of proceeding with these cases. In 1914 Holt[193] reported fifty-seven cases with a mortality of 55 per cent. Of these, twenty-eight patients were operated on with fourteen deaths, and twenty-seven were treated medically with seventeen deaths. Ladd's[203] table shows that prior to 1915 the mortality with the Rammstedt operation was 33 per cent.; during 1915 it was 28.5 per cent.; during 1916 it diminished to 15.3 per cent.; while Kerley[206] quotes the operative mortality for the year of 1915 as being 50 per cent. Holt[211] reports a mortality of 58 per cent. among patients treated medically from 1901 to 1911, and 58 per cent. among patients treated surgically. From 1912 to 1914 the mortality among the medically treated was 57 per cent., among the surgically treated 43 per cent. In 1915 and 1916, the mortality among medically treated patients was 50 per cent., and 61 patients treated by Rammstedt's operation gave a mortality of 23 per cent. In 1916 Dr. Sedgwick[208] reported forty-six cases with two deaths. All except two patients were treated medically. One patient in extreme condition when first seen died under medical treatment. One patient operated on died and one survived. Proper medical treatment supported by proper operative treatment in selected cases, he believes will give the best results. Dr. DeBuys also believes that no case should be considered surgical until it has been proven nonmedical. Abt believes that if the baby loses more than one-third its body weight, it has reached the danger point and should be subjected to operation.

The fact that the tumor may absorb and leave the lumen open does not contraindicate the necessity for operation, Richter[200] states; the fact that it does not disappear quickly enough is the essential feature. Of twenty-two cases in which he operated, nineteen were typical posterior gastro-enterostomies, with but two deaths which were attributable to faulty technic. The surviving child gained continuously and presented in every way the picture of a normal child. Of 101 cases observed by Strauss,[202] sixty-five patients were treated surgically with three deaths and thirty-six were treated medically with no deaths. In sixteen of the thirty-six medically treated patients, the stomach emptying time was normal. Peristalsis started high on the stomach, forcing the food through and a hyperperistalsis was noted in the entire intestinal tract. In the other twenty medical cases characteristic contractions of the pylorus were seen. He calls attention, however, to the complete and immediate relief afforded by operation. According to Downes[195] under the most favorable condition and in the

best hands, the cases treated medically are long drawn out, always in doubt, and the knowledge ever present that without the slightest warning the baby may go into collapse, and die even though its progress has been favorable.

A splendid article appeared in May, 1917, by Emmett Holt,[211] in which he describes the symptoms and gives the results of the treatment, both medical and surgical, in his series of cases. He feels that there is a congenital hypertrophy of the pylorus with a superimposed spasm in all cases, and that the palpable tumor is usually present. He follows the medical treatment for two or three weeks in mild cases, but recommends the Rammstedt operation as soon as the diagnosis is made in severe conditions. Dr. Kerley[206] asserts that the presence of a palpable tumor demands immediate surgical intervention, and places the mortality at not over 5 per cent. if the Rammstedt method is employed when the child has not been vomiting over two weeks. LaFetra[212] finds his indications for operation in progressive loss of weight together with absence of any food residue in the stools. As long as there is evidence of any food passing into the intestines, he believes one is justified in carrying out medical treatment. The palpable tumor may not necessarily mean hypertrophy. Reuben[192] published a table showing 94 per cent. recoveries with medical treatment and 80 per cent. recoveries with operative treatment. He favors surgery if the infant weighs less than 6 pounds and does not respond to medical care within from seven to ten days, and if the loss of weight is rapid, from 8 to 16 ounces a week, or if the vomiting persists in spite of all efforts.

Among the successful results following posterior gastro-enterostomy Lewis and Grulee[213] published the report of a case which came to necropsy 256 days after operation and in which the opening had evidently been working perfectly. The child had gained 4 pounds and 5 ounces. However, the Rammstedt procedure and its modifications are giving far better results. Rachford[199] reports three successfully operated cases, the first by gastro-enterostomy, the second and third by a typical Rammstedt. In the first case the rapid passage of food through the artificial stoma interfered, and was likely always to interfere, with the perfect functioning of the gastro-intestinal tract, the end result being nothing like as good as that obtained by the

211. Holt, L. Emmett: Hypertrophic Stenosis in Infants, J. A. M. A. **68**: 1517 (May 26) 1917.

212. LaFétra, L. E.: Accidents and Diseases During Early Weeks of Life, Arch. Pediat. **33**:401 (June) 1916.

213. Lewis, Dean, and Grulee, C. G.: The Pylorus After Gastro-Enterostomy for Congenital Pyloric Stenosis, J. A. M. A. **64**:410 (Jan. 30) 1915.

Rammstedt method where the pyloric tumor mass not only disappeared but the pylorus resumed its normal physiologic function. In discussing this later method, Ladd[203] makes the point that it is when the tumor is hard that the mucosa separates from the mucsular layer easily, and the muscle splitting operation is very successful.

Dr. Strauss[202] asserts that by his method of pyloroplasty, a mortality of 30 per cent. with the Rammstedt is reduced to 4 per cent. He makes an incision about 1 inch in length and through it a ribbon-shaped hook is inserted, and the tumor drawn out. By resecting one-half of the circumference and one-half of the length of the pylorus and pyloric antrum, he reconstructed the resected half by two superimposed free fascial transplants and acquired a perfectly normal functioning pylorus. When the mucous membrane is separated from the inside he separates Meisner's from Auerbach's plexus, which accounted for the paresis of the pylorus. By cutting down to the mucosa, and shelling it out, he reduced the folds of the infolding membrane, and produced a paresis of the spastic pylorus. By cutting away the inner circular layer or utilizing it as a plastic flap, he reconstructed the hypertrophied musculature to its normal thickness. The advantages of this pyloroplasty over posterior gastro-enterostomy, he believes to be the smaller incision necessary, vastly smaller amount of shock, one third the time, every form of pathologc condition of congenital pyloric stenosis may be covered, a normally functioning pylorus is secured, and the normal relation of stomach to bowel is preserved. The Rammstedt gives relief in younger children when the tumor is small but in older children it is necessary that the mucosa should be shelled out. In the usual type of case he gives 1 ounce of physiologic sodium chlorid solution by rectum every three hours following operation. After the child is awake, it is fed 1 dram of breast milk every two hours with 1 dram of water in the interval.

Keefe cuts a crescent shaped piece from each side of the original incision into the tumor, then fastens an omentum overflap with one stitch and drops the pylorus back, the entire operation employing but four minutes. He considers Strauss' procedure more complicated than is necessary, while Weller Van Hook styles it not only unnecessary but mischevious.

Haggard[201] employs local anesthesia, 0.1 per cent. procain, in this operation, considering it safer, more pleasant and more satisfactory than ether. He gives no narcosis.

Twenty-six patients attended by Kerley[206] ranged in age from three to sixteen weeks at the time of operation. Postoperative vomiting occurred in only nine cases; all these patients recovered. Only

four of the twenty-six patients died, three of whom had vomited for weeks and were in very bad condition when first seen. He advises that immediately after operation the patient be wrapped in a warm blanket, surrounded by hot water bottles, a hypodermoclysis of 120 c.c. of physiologic sodium chlorid solution given and the head lowered from one to two hours to prevent aspiration of mucus into the larynx. Epinephrin is administered if the child's condition is poor, and dilute whiskey is given every four or five hours when necessary. One and one-half hours after operation 10 c.c. of water is given, later barley water and breast milk, the patient being allowed to nurse by the eleventh or twelfth day.

The medical treatment for pyloric stenosis in most general use is that outlined by Sedgwick.[208] It must be persistent, well planned and carefully carried out, and consists of lavage before feeding, breast milk every four hours if attainable, gavage when necessary, and external heat to maintain body temperature. Large doses of chloral hydrate by rectum are of value in some cases. After observing the striking effect of thick cereal feeding in six cases of neurotic vomiting, Sauer[214] tried the same mode of treatment in pyloric stenosis. In eleven of the twelve cases reported the vomiting soon ceased. Farina is cooked in breast milk and fed very thick. A great deal depends on the manner of preparation and mode of feeding. Sauer is convinced that it should have a very thorough trial before surgical treatment is resorted to.

The use of atropin in cases of pure pylorospasm is advocated by Haas.[198] He gives a five minute feeding every two and one-half hours, preceded by one drop of 1:1,000 solution of atropin sulphate in 1 dram of water, the dose to be increased daily by one drop at each feeding until physiologic effects are observed. The largest dose that he has given was 1/16 grain in twenty-four hours. As indication of the patient's tolerance for atropin he points out that pallor and not flushing denotes danger, and that some patients who tolerate small doses poorly, take large doses well.

In summing up one must remember that the results reported from surgical treatment are those attained by exceptionally clever operators with a host of experience in this field. If the records of the average surgeon were published the results might be very much less gratifying. Likewise, it is not likely that the percentage of cures reported from medical treatment as carried out by its most capable and experienced advocates would in any way represent that resulting from the vacillating methods of the average medical man.

214. Sauer, L. W.: Use of Thick Farina in the Treatment of Pyloric Stenosis, Arch. Pediat. **35**:385 (July) 1918.

CONGENITAL HEART DISEASE

By comparison of clinical and postmortem findings in forty-eight cases, Dunn[215] finds the essential diagnostic features of the various congenital cardiac lesions to be as follows:

1. A case showing cyanosis with enlargement of the cardiac dullness or palpable thrill, or both, is one of pulmonary stenosis. If the baby dies shortly after birth, the most probable lesion is pulmonary stenosis alone. If the baby survives early infancy, or lives on into childhood, the pulmonary stenosis is probably associated with some other lesion. If the murmur is notably transmitted into the vessels of the neck, or if the humming top murmur is present, the additional lesion is probably open ductus arteriosis. If the murmur has neither of these characteristics, the complicating lesion is probably defective interventricular septum.

2. A case showing a murmur and enlargement without cyanosis, is probably one of defective interventricular septum. If the murmur is not transmitted into the neck this lesion exists alone. If the murmur is so transmitted, or if the humming top murmur is present, the lesion is probably combined with open ductus arteriosus.

3. A case showing a murmur, without either cyanosis or enlargement, especially if the murmur is markedly transmitted into the vessels of the neck, or if it extends into diastole, is probably one of open ductus arteriosus alone. If the murmur is of the humming top variety, extending throughout the cardiac cycle, one may make a diagnosis of this lesion with almost absolute certainty.

The prognosis does not vary with the character of the lesion except in two forms: pulmonary stenosis alone and open ductus arteriosus alone. In all other forms, there is evidence that the patient may survive, and even live to adult life. The prognosis in babies cyanotic at birth, is grave, as many of them have pulmonary stenosis alone, and die within a very short time. In those who survive, the pulmonary stenosis is probably combined with some compensating lesion, and is that of congenital cardiac disease in general. In cases which show the clinical picture of open ductus arteriosus alone, the prognosis is very good.

A case of endocarditis is reported by Tuley and Moore[216] with necropsy findings which they believe to be of congenital origin.

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215. Dunn, Charles H.: Contribution to the Diagnosis and Prognosis of Congenital Heart Disease, Am. J. Dis. Child. **8**:185 (Sept.) 1914.

216. Tuley, H. E., and Moore, J. W.: Report of Case of Congenital Endocarditis, Am. J. Dis. Child. **13**:426 (May) 1917.

Bass[217] reports a case of heart block, and reviews the literature on the subject. He concludes that in childhood it occurs in rare instances in hearts affected by congenital defects, and that until postmortem examination is made we must remain ignorant of the exact relationship of the cardiac irregularity to the cardiac malformation.

After reviewing all of the available literature pertaining to the condition Weber[218] finds only three cases in which the clinical manifestations of congenital heart disease have disappeared during the progress of the general growth and development of the child. All the patients were boys. The first child was seen at fourteen months of age. At that time there was a very loud systolic murmur with its maximum intensity over the fourth left intercostal space, close to the sternum, but audible also over the whole precordial region, and indeed, over most of the chest both back and front. It was accompanied by a thrill near the sternum and a diagnosis was made of congenital aperture in the interventricular septum. When the boy was 2 years old, the murmur was about the same as before, very loud and universal, but when he was 5 years old, there was absolutely no murmur to be heard. At the age of 10 years, there was no objective evidence of the presence of any heart lesion at all. The other two cases were similar in every respect, the murmur disappearing when the patients were 6 years of age.

Moffett and Neuhof[219] have reported a case of pure dextrocardia with patent ventricular septum. They accompany the report with a bibliography of 126 cases of pure dextrocardia, covering everything from the years 1649 to the present time. A case of pure dextrocardia is also reported by Morgan[220] and confirmed by necropsy. The child was cyanotic from birth, had rapid respiration and a systolic murmur could be heard at times. The left lobe of the thymus was enlarged, but all other organs were found in their normal positions. The child died at nine months of age.

A form of congenital cyanosis is described by Grandjean,[221] which showed at necropsy a uniform contraction of the pulmonary artery

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217. Bass, Murray H.: Heart Block and Congenital Heart Disease in Childhood, J. A. M. A. **70**:287 (Feb. 2) 1918.

218. Weber, F. P.: Can the Clinical Manifestation of Congenital Heart Disease Disappear with the General Growth and Development of the Patient, Brit. J. Child. Dis. **15**:113 (April-June) 1918.

219. Moffett, R. D., and Neuhof, S.: Congenital Dextrocardia, with Patent Interventricular Septum, Am. J. Dis. Child. **10**:15 (July) 1915.

220. Morgan, H. J.: Congenital Dextrocardia with Patent Ductus Ovale, Necropsy at Nine Months, Am. J. Dis. Child. **12**:233 (Sept.) 1916.

221. Grandjean, M.: Congenital Cyanosis without Auscultatory Signs, Ann. de méd. et chir. inf. **18**:495 (July 15) 1914.

and an equal thickness of the walls of the two ventricles, no thrill or murmur and no vibration of obstructed blood current were noticeable. Cyanosis was complete and crises of suffocation occurred. Auscultation gave almost no signs but an enlargement of the cardiac area. He considers the prognosis grave, the child having slight resistance to infections. Another case is reported by Morse,[222] that of a child 4 months of age with lips, cheeks and nails blue, and at times blue all over. At 4 years it was cyanotic, had marked clubbing of the fingers and toes, bulging of the precordia and enlarged heart. The hemoglobin ranged from 120 to 140 per cent. At the age of 9 years the child died suddenly, and the necropsy showed complete congenital atresia of the pulmonary artery; congenital defect of the interventricular septum; persistent ductus arteriosus; acute vegetative endocarditis; thrombosis of ductus arteriosus; patent foramen ovale; dilatation and hypertrophy of the right ventricle. The heart was greatly enlarged, weighing 130 gm. A single arterial trunk rose at the base of the ventricles anterior to the auricles.

A case in which the aorta was completely closed for a distance of 2 c.c. has been reported by Gauss.[223] Beyond this point it dilated and reached its normal diameter at the juncture of the ductus arteriosus.

Forschbach and Kolvczek[224] report the findings in two patients, 10 and 17 years of age, respectively. During prolonged inspiration and the Valsalva test, the systolic murmur dwindled or disappeared and the roentgen-ray shadow showed a flattening of the second arc of the heart's left outline, demonstrating a reduction in the pressure in the aorta under the influence of the increased alveolar pressure.

In a case of Van Tilburg's,[225] a previously healthy young lady with patent ductus Botalli developed pulmonary tuberculosis, fatal in two years. The puzzling auscultation findings were explained by a thrombus obstructing the opening duct and hanging loose in the pulmonary artery. Simons reports another case of ductus arteriosus in a man of 21 years of age with no history of rheumatism. A continuous murmur was heard with a thrill in the second and third left spaces and a localization of the murmur in both interscapular

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222. Morse, J. L.: A Case of Congenital Heart Disease with Demonstration of Specimen, Am. J. Obst. **72**:558, 1915.

223. Gauss, H.: Congenital Obliteration of Aorta, Am. J. Dis. Child. **12**: 606 (Dec.) 1916.

224. Forschbach, J., and Kolvczelk, M.: Symptomatology of Patent Ductus Arteriosus or Botalli, München. med. Wchnschr. **63**:1617 (Nov. 14) 1916.

225. Van Tilburg, J.: Congenital Heart Defects, Nederl. Tidschr. v. Geneesk. **1**:1397 (May 18) 1918.

226. Simons, Irving: Ductus Arteriosus, J. A. M. A. **62**:35 (Jan. 3) 1914.

regions. The cardiac hypertrophy was left sided. With the exception of a period of two weeks there had been no discomfort due to the heart, and he lifted and ran upstairs with as little dyspnea as a normal man. The author includes measurements and an illustration.

In a case of patent foramen ovale seen by Finley and Martin[227] the conditions reported at necropsy were: hypoplasia of eyes with corneal opacity, coloboma and congenital cataract, ventral hernia, fetal lobulation of spleen and kidneys, patent foramen ovale, catarrhal bronchitis, with early pneumococcic bronchopneumonia. Another case reported was a congenital stenosis of the pulmonary tract with atresia of the orifice, incomplete interventricular septum, patent ductus arteriosus and patent foramen ovale. Extensive pneumococcic bronchopneumonia developed and death occurred at three months. In one case terminating fatally at two months necropsy showed congenital stenosis of the pulmonary tract, with a defect of the interventricular septum at the pars membranacea, widely patent foramen ovale and duplication of the vena cava superior.

In the third case congenital syphilis was disclosed at necropsy with patent foramen ovale and patent ductus arteriosus and venous congestion of the organs. The child died when one month old.

Kilduffe[228] gives a very complete history, physical examination, and necropsy report in a case of congenital absence of the lower half of the interauricular septum which gave no serious symptoms until just before death and was unrecognized until necropsy. Hess reports a case of complete atresia of the right auriculoventricular orifice with absence of the tricuspid valve. The patient died when eight months of age and at necropsy, in addition to the above conditions, he found an atrophic right auricle, a patent ventricular septum and ductus arteriosus.

McCulloch[229] reports the electrocardiographic studies in twelve cases of congenital heart disease with necropsy reports. He finds that congenital malformation of the heart does not produce a distinctive electrocardiogram, but the type of curve depends on the secondary effects on the heart chambers. This effect is chiefly on the right side of the heart, and malformation associated with a secondary relative right ventricular hypertrophy, produces electrocardiogram with a deep S wave in lead one, and a high R wave in lead three. Malformations

227. Finlay, L., and Martin, W. B. M.: Case of Congenital Heart Disease, Glasgow M. J. **86**:1 (July); 84 (Aug.); 140 (Sept.) 1916.

228. Kilduffe, R.: Interauricular Septum; Case of Congenital Absence of Lower Half, Am. J. M. Sc. **147**:880, 1914.

229. McCulloch, Hugh: Electrocardiographic Studies of Congenital Heart Disease, Am. J. Dis. Child. **12**:30 (July) 1916.

not producing right ventricular hypertrophy fail to cause a characteristic change in the electrocardiogram.

THYMUS GLAND

Schridde,[230] in reporting postmortem examinations on fifty-nine stillborn infants, says that the lymph nodes in the spleen and intestines are not visible. The tonsils are visible, but it is difficult to determine the size. He states that the thymus has a normal range of variation of from 3 to 14 gm. in weight, according to the length of the fetus, while Wernstedt believes that the thymus from 20 to 30 gm. in weight should be regarded as normal. LeBoutillier,[231] however, believes with Holt, that 7.4 gm. is the normal average weight at birth. The gland increases or decreases in size according to the state of nutrition of the child, gradually increasing in size for three or four years, then decreasing gradually until puberty, when, as a rule, it disappears. Thymus enlargement causes pressure on some of the important organs in the structure of the body. Some are thin and spread out, while others are thick, firm and unyielding, almost filling the entire space between sternum and vertebra. The various conditions caused by enlarged thymus he believes to be tracheal stenosis pressure by the gland on the circulatory system and nerves, status lymphaticus, laryngospasm and dyscrasias. He reports a case of death of a new-born believed to be due to enlarged thymus. The death was sudden and without premonitory symptoms. The thymus was very large, weighing 65 gm. and covering the entire anterior surface of the heart. There was no tracheal stenosis and death was believed to have been caused by direct pressure on the heart and its ganglions.

A case of thymus death in a new-born has been reported by De Forest[232] where, notwithstanding every effort at artificial respiration, no air could be made to enter the lungs. The heart beat for twenty-five minutes after delivery but gradually ceased. Necropsy showed a complete atelectasis of both lungs and a tracheal obstruction due to an enlarged thymus gland. The trachea was found to be quite flat, the anterior wall compressed against the posterior. The atelectasis was not due to lack of normal development of the lung tissue but to mechanical obstruction. The foramen ovale was also widely opened.

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230. Schridde, Hermann: Der Angeborene Status Thymolymphaticus, München. med. Wchnschr. **61**:2161, 1914.

231. LaBoutiller, Theo.: Hypertrophy of the Thymus and Thymus Death with Reports of Cases, Arch. Pediat. **32**:322, 1915.

232. DeForest, H. P.: Thymus Death in a Young Infant, Am. J. Obst. **69**:693, 1914.

The chief symptoms of enlarged thymus as recognized by Herrick[233] are those of obstruction to respiration, but he alleges that much of the trouble may be due to pressure on other organs than the trachea. The respiratory difficulty may be a mild stridor or a severe dyspnea with fatal termination. However, the immediate cause of death may be pressure on the vagi, the vessels and the heart, as well as on the trachea. In one case mentioned there was good reason to believe that there was direct pressure on the right auricle. The thymic patient is usually well nourished but pale and pasty with a negative tubercular history. Extirpation is both dangerous and uncertain, the roentgen ray being much the safest and most satisfactory treatment. Six cases, the patients ranging in age from four weeks to three years, are quite fully reported. In two cases symptoms appeared one week after birth, in one at two weeks, in one at two and one-half months, in one at one year, and in three at three years. Two patients died with practically none but symptomatic treatment, one patient lived but in very bad condition, under the same treatment, while three patients were treated with the roentgen ray and apparently are well.

Microscopic findings and other minute data regarding the thymus from sixteen cases are given by Hammar.[234] He compares his findings with data from an equal number of apparently healthy children killed in accident or losing their lives from external causes. In no respect did he find any dissimilarity in the conditions of the compared thymuses, and he concludes that the thymus is not responsible for sudden death, even in those typical cases known as "thymus death."

Palancar and de Arcante[235] declare that microscopic findings are more conclusive as testimony than the mere size and shape of the thymus in cases of sudden death for which the thymus seems to be responsible. Their conclusion is that the thymus in certain cases may exert a toxic influence independent of any mechanical action. With hyperplasia of the thymus there may be perverted functioning which may entail gravest accidents. In their series the lymphatic system seems to be normal otherwise. This suggests that there is not the connection between the lymphatic apparatus and the thymus which some assume. The mechanism may not always be the same in the cases of sudden thymus death, toxic factors intervening in some and mechanical factors in others. The microscopic findings in the case

233. Herrick, J. F.: Report of Cases of Enlarged Thymus in Infancy, Surg., Gynec. & Obst. **22**:333 (March) 1916.

234. Hammar, J. A.: Microscopic Analysis of the Thymus in Sixteen Cases of Sudden Death from External Causes, Svenska Läk.-Sällsk. Handl. **17**:867, 1916.

235. Palancar, J., and de Arcante, L. R.: Thymus Death, Arch. espanoles de Pediat. **2**:208, 1918.

reported showed a remarkable number of subpericardial and subcapsular ecchymoses, one apparently in every terminal arteriole. The article is illustrated.

An exhaustive review of the literature on the thymus gland is made by Lerch,[236] covering the physiology and histology, pathology, symptomatology and treatment. He asserts that severe chronic disturbances may be expected after thymectomy in children at the end of the second year, which will result in death at puberty. The operation should not be performed during the time of greatest development of the glands. The blood, temperature or metabolism are not influenced by the operation. Children with hyperplastic thymus have not alone to suffer from the pressure of the organ on the trachea and vessels but also from the intoxication which gives rise to the lymphatic chlorotic constitution. In infantile marasmus with atrophy of the thymus, the condition sometimes responds to thymus extract. The function of the thymus is of importance to stimulate growth in childhood. The form and position of the organ remains the same throughout life.

Delavan[237] uses the roentgen ray in the diagnosis of enlarged thymus and finds the results valuable and interesting. Careful instructions should be given to clinical assistants to the end that they be warned as to the dangers of operating on patients having enlarged thymus, and that they become competent to diagnose such cases when presented. Without doubt the use of the roentgen ray before operation would result in the saving of life.

Among other cases of hyperthymatism Sylvester[238] reports one in a child 5 months of age. The family history was negative. The child was breast fed, had no digestive disturbance, except a difficulty in nursing owing to its rapid breathing and had gained slowly but consistently. A tendency to constipation was noted. The mother thought the child had suffered from a growth in the throat since birth because she could not breathe through her nose. When nursing she would suck once or twice, then gasp for air. Four roentgen-ray treatments were administered, of five minutes duration each, on succeeding days, and the symptoms practically disappeared after the fourth exposure. The child remained free from symptoms for four months, when the asthmatic breathing reappeared. She was again treated and a complete recovery apparently effected.

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236. Lerch, Otto: Contribution on the Thymus, Med. Rec. **85**:648, 1914.
237. Delavan, D. B.: Employment of Skiagraphy in Diagnosis of Enlargement of Thymus Gland, Med. Rec. **86**:665, 1914.
238. Sylvester, P. H.: Diagnosis of Enlarged Thymus and the Symptomatology of Hyperthymatism, Boston M. & S. J. **170**:528, 1914.

The literature is again reviewed by Oliver[239] and a case is reported of cystic thymus in a syphilitic new-born. Syphilis of the thymus manifests itself primarily by degeneration of the lymphocytes and Hassall bodies with hypertrophy of the reticular epithelium and hyperregeneration of their concentric corpuscles, this process giving rise to cystic cavities by the dilatation, and fusion of the degenerating Hassall bodies.

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239. Oliver, Jean: Syphilitic Disease of the Thymus in Infants, Am. J. Dis. Child. **13**:158 (Feb.) 1917.

CHILD WELFARE*

Child welfare is an essential part of a general program in public health and preventive medicine and cannot be separated from it. It is particularly closely associated with housing and food production and supply, for children require food not merely for sustenance but for growth.

Children, as the weakest members of the community, feel first and most heavily the effect of all unfavorable hygienic, social and economic conditions. The war has increased infant and child mortality, and in some countries the birth rate has been lowered more than half. The health of many children who have survived has become greatly impaired by reason of improper food and neglect. Furthermore, the war and recent epidemics have enormously increased the number of dependent children, especially in the devastated countries.

A reduced birth rate and an abnormal sickness and death rate may be expected to continue for several years to come; a reduced birth rate, owing to death of potential fathers, to social and economic conditions which have affected the health of potential mothers, and to many other factors; an increased sickness and death rate, because of the scarcity and high cost of food, and general hardship and privation which favors the spread of epidemics and lowers resistance to all forms of disease. A world wide child welfare campaign is, therefore, one of the most urgent needs of the day, both from a humanitarian and economic standpoint; as the children are the nation's greatest asset.

The pressing problems now are:

1. To save the infants yet to be born and to promote their healthy development.

2. To restore the health and make possible the normal growth and development of children who are now suffering from disease or defective nutrition and to safeguard the health of those whose nutrition has not yet suffered.

3. To do something for the immediate needs of dependent children.

Experience has shown that no efforts in public health work produce more immediate or far reaching results than those which are put forth in child welfare.

The work relating to child welfare may be divided into six parts, corresponding to the period of child life to which it is directed:

First.—Eugenic consideration affecting the prospective parents.

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* Report from International Congress.

Second.—The prenatal period in which the child is saved through care for the mother.

The importance of this may be appreciated by the fact that even in the highly civilized countries in peace times

(a) The still-births are equal to between 4 and 5 per cent. of the living births.

(b) That as a result of premature birth many infants born die in the first weeks of life.

(c) That other causes of the high mortality during the early weeks are due to conditions affecting the mother.

Methods of prenatal care will necessarily differ in urban and rural communities and also be modified by race, customs and environment. The essential features must be the supervision and education of the expectant mother by trained public health nurse, midwife, or health visitor from an early period of her pregnancy, aided when possible by the advice and assistance of a physician. The work is greatly facilitated by voluntary registration of pregnant women, which should be encouraged everywhere, and it should be connected closely with maternity and child welfare centers.

Success in any effort in child welfare is largely dependent on the intelligent cooperation of both parents; no plan of education, therefore, is complete which does not include the father. This is particularly important in the prenatal period and in the period of infancy.

Third.—Obstetrical care, which should include the services of a nurse as well as those of a physician or midwife. Proper care saves the child from accidents during delivery; saves the mother from immediate and remote dangers incident to her confinement; greatly increases the chances of successful breast feeding and prevents much blindness.

Fourth.—Birth to school age. This period is sometimes divided into infancy — including the first year or possibly two years — and a preschool age, extending from this time up to the fifth or sometimes the sixth year.

The important concern during the first two years is the child's nutrition; and the essential thing is that the child be kept under continuous observation and supervision. Everything possible should be done to promote breast feeding, as by far the most important means of insuring vigorous growth and development and preventing infant mortality. At first, the supervision must be close, the infant being seen in many cases every week or even oftener. The visits will be less frequent as the child grows older, being made at least monthy after six months, and every two months up to the end of the second year.

Success during this period depends almost entirely upon our ability to educate the mother and to arouse and maintain her interest in her child's growth and progress.

The principal methods are:

1. Group instructions and individual advice in a central place known as a "milk station," a child welfare station, a "consultation," etc., and

2. Home visiting by public health nurses or health visitors.

Both methods have their place and should usually be combined, certainly in urban communities.

The problem of infancy up to the school period is much the same and can be carried on with the same organization — a quarterly visit to the home or by the child to the central stations and a full medical examination once a year should be the minimum. Much better results would be obtained if the observations were made more frequently. The above requirements should be for every healthy child. Those who are delicate or ill should have special medical attention.

By these means the beginning of organic disease may be detected, physical defects and deformities prevented or corrected, and a general supervision maintained over the child's diet and general hygiene.

Records should be kept of all abnormal conditions and any serious illness, also of height and weight, which will in themselves furnish a good guide as to health progress.

Permanent institutional care for infants and young children should be discouraged on account of the almost insuperable difficulties in maintaining nutrition in infancy under these conditions, and because of the great susceptibility of young children to infection, preference should be given to placing such children in suitable families. All creches, day nurseries, and the like should be under close medical attention.

Fifth.—In the School Period. From the sixth to the fourteenth or sixteenth year, the problem is to secure normal growth and physical and moral development, to recognize and correct defects which interfere with these, and to reduce to the minimum the risks of contagious disease. The school physician, the school nurse and the school teacher all have important functions. The school, furthermore, offers an opportunity, the value of which is only beginning to be appreciated, to interest, instruct and train the child himself in health matters. It should be the duty of schools to teach health as much as to teach reading and writing. Health work for the school children is inextricably bound up with the problems of general education, and all teachers should receive training which would qualify them to teach the simple facts of health and personal hygiene.

An important kind of health education which can be carried on in the school is the instruction of girls from the age of 10 years upward in the rudiments of infant care. This has been proved to be valuable in preparing them to undertake the responsible charge of their younger brothers and sisters, and what is even more important, in assisting to prepare them for the future duties of motherhood.

The child's mind is virgin soil for health instruction of every sort. It is not necessary at this period to clear away the rubbish of prejudice, superstition and habits based upon ignorance, which make health education so difficult and often so futile in adult life. Scientific facts in regard to food, health habits, hygiene and the spread and control of disease can, if presented by writers and artists who know how to reach the child's mind, be made both interesting and attractive. Why should not the stories of the conquest of smallpox, malaria and yellow fever be made as thrilling as the conquests of Alexander or Napoleon?

This instruction should be begun with children of eight or nine, for early impressions are the most lasting. The bare facts of hygiene or physiology as presented in school textbooks make little impression that is permanent. Their acquisition is made work. Health instruction should and can be made play. Health may even be made a competitive game. If the school children can be properly instructed in health matters we need have no anxiety about the adults of the next generation.

Progress in the pupil's health is best shown by the weight chart; thus an incentive can be furnished for the observance of health rules which are taught.

Health records, at least a record of weight and growth, should be kept during school life. In an ideal scheme, a medical examination should be made once a year of every school child, and those who are found to be physically below normal or who do not make a normal gain in weight should receive special attention and supervision during their school life.

Sixth.—Industrial period from the fourteenth to the eighteenth year. In connection with legislation relating to child labor, the following fundamental principles should be embodied:

(a) No child should be allowed to enter industry without first passing a physical examination by a competent physician, showing that he is able to perform the work intended.

(b) No child should be permitted to continue in any form of work which prevents his normal growth and physical development as determined by accepted standards, and periodic examinations made up to the age of 18 years.

Any intelligent action must be based upon an exact knowledge of conditions. Therefore, the first step thould be a survey.

After a survey has been made in any activities which are contemplated there are certain fundamental principles which should be kept in mind if the work is to be effective and anything permanent accomplished.

(1) Local customs, climatic conditions, diet and other things on which are based the habits of the people should be carefully considered and appreciated.

(2) All local health habits and racial customs, which are good, should be accepted and used as a foundation on which to build.

(3) It is impossible to formulate beforehand a plan of procedure which can be followed closely. Any plan adopted must have great elasticity and be modified by circumstances as they arise in the course of the work.

(4) Local agencies, organizations and individual workers should be developed and utilized so far as possible.

(5) The utmost tact is necessary to adopt knowledge and experience gained elsewhere to work which is instituted under new conditions and among new people.

(6) No plan of work should be made so complex or so expensive that it cannot be duplicated or extended by a simple organization elsewhere.

(7) Success depends much less on the program adopted than on the personnel which will put it into effect.

(8) To carry out effectively any scheme of child welfare such as has been outlined, it will be necessary to provide material assistance in a considerable proportion of cases.

Dr. Lucas presented an additional detailed program for child welfare work in the community. It was recorded with the above program.

The Council at Cannes adopted, among its final resolutions:

(1) That the control of typhus fever be at once undertaken as an emergency relief measure.

(2) That the wide extension and development of child welfare work be selected as the first important constructive activity.

(3) That the training of schoolchildren in all grades by thoroughly qualified teachers in the subject of personal and general hygiene and the inculcation of proper health habits during school life are perhaps the most important undeveloped measures for permanently improving the health and contributing to the welfare of the people.

Finally, a communication from the acting secretary of the League of Red Cross Societies, dated June 2, 1919, from Geneva, Switzerland, states that "It is now confidently believed that the League of Red Cross Societies, now prepared to engage in a concerted and intelligent effort in behalf of the health and welfare of the world, will prove to be one of the most significant of modern institutions for the advancement of civilization. The series of recommendations will form the foundations for the practical work of amelioration which the League of Red Cross Societies is about to undertake on a world embracing scale."

REPORT OF THE CHILD WELFARE SECTION OF THE COMMITTEE OF RED CROSS SOCIETIES AT THE CONFERENCE HELD AT CANNES, APRIL 1, 1919

The Conference was called at Cannes, April 1, 1919, with Professor Roux, Director of the Institute Pasteur, chairman of the meeting.

The Child Welfare section was composed of the following representatives from the various countries: Sir Arthur Newsholme, chairman; Dr. Henry Kenwood and Dr. Truby King, representing England; Professors Baduel and Valagussa, representing Italy; Professor Pinard, Professor Pehu and Dr. Armand Delille, representing France; Dr. L. Emmet Holt, Dr. W. Palmer Lucas, Dr. Samuel McClintock Hamill, Dr. Fritz B. Talbot, secretary, and Miss Lillian D. Wald representing Ameria.

CURRENT PEDIATRIC LITERATURE

PATHOLOGY AND BACTERIOLOGY

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